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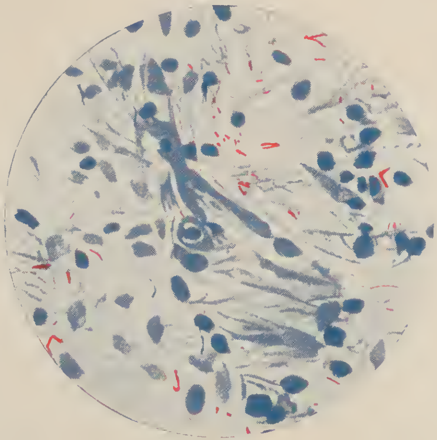
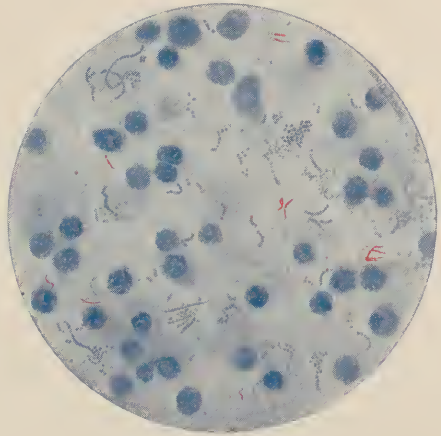


A. Tubercle Bacilli in Sputum



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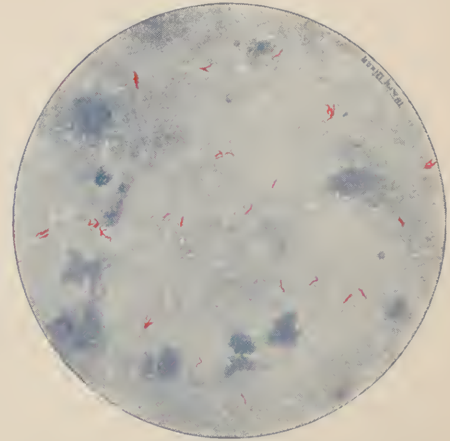

C. Tubercle Bacilli in Meningeal Fluid

Descriptive Text, Page xviii.

Handbook *of* Tuberculosis

for

Medical Students and Practitioners of Medicine

by

JOHN RITTER, PhG., M.D.

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One Colored Plate (frontispiece) Showing Stained Specimens with Tubercle Bacilli Under the Microscope, and Sixty-One Illustrations Throughout the Text

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CHICAGO, 1923

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This Volume

is affectionately inscribed to the earnest medical student in quest of accurate knowledge concerning the many intricate problems in tuberculosis, and to the busy practitioner who wishes to refresh his memory about the various tuberculous disorders which may confront him in his usual daily labor, it is respectfully dedicated by the

AUTHOR.

INTRODUCTION

The compilation of this volume is based upon lectures, recitations, quizzes and clinical demonstrations given for many years at Rush Medical College to students of medicine and to medical practitioners who were especially interested in the problem of tuberculosis. In addition, abstracts which have been prepared from papers on tuberculosis topics which were read before the National Tuberculosis Association and from addresses delivered before various tuberculosis and health conferences furnish a part of this volume. Further, in the second part of the book—the section on Clinical Tuberculosis—appear many lectures and papers on Tuberculosis and its Complications delivered or read before my classes by well-known specialists in their respective branches, to whom due credit is given and mention made. These lectures and papers have been either abstracted or appear in their entirety in that section.

A perusal of this volume will convince the reader that in many respects it differs very much from the general text or hand books of tuberculosis. Having taught for years the principles underlying physical diagnosis, I have learned one thing, namely, that these principles as they are imparted to students are not always well understood and not only that, often times, even after years of practice, these principles of physical diagnosis are still somewhat hazy and vague.

The principles of physical examination like anything else in medicine which is to lead to success, can only be acquired by constant and diligent observation, repetition and study, and this requires time. For a number of years I have taught that a mastering of the principles of physical diagnosis requires the careful examination of hundreds of chests or perhaps a thousand, but I have long since modified this teaching and I now say that when you have made many thousand careful chest examinations, you may, perhaps, have acquired a fairly good knowledge of these physical findings, provided you have thoroughly understood and were able to interpret correctly the signs observed and the sounds heard.

As a thorough knowledge of physical diagnosis is most essential to an understanding and a proper interpretation of the sounds

produced within the chest cavity (and this applies particularly to pulmonary tuberculosis), it occurred to me that a constant reminder of the findings in the normal chest is absolutely necessary, and so I have drawn parallel lines between which I constantly endeavor at all times to draw a comparison—the normal with the pathological. It has been my privilege to observe that by such a method the student is better prepared to master and to understand the different chest findings, that is, by comparing the abnormal with the normal, right on the spot, rather than to refer only occasionally to normal findings; this plan is followed throughout the entire section on clinical tuberculosis.

The study of tuberculosis and of the tuberculosis problem in general as the physician encounters it in his daily work may be viewed from a threefold angle, namely, the social, the economic and the medical aspect. Both the social and the economic aspect belong more appropriately to the domain of the social worker, the philanthropist, the charity organizations and to the various antituberculosis organizations and movements. Although the physician must be vitally interested in the whole problem of tuberculosis, in the social as well as in the economic aspects, his chief interest, usefulness and influence lie in combating the disorder from the medical standpoint.

As this manual is mainly of interest to students in medicine and to physicians, I will not consider the problem other than from a medical standpoint, leaving the social and economic aspects to their proper domain. The medical aspect of this important and interesting problem in tuberculosis may for convenience of study be presented in three distinct and separate parts, namely, part one, which considers all those questions purely of an academic nature, part two, the elucidation of clinical tuberculosis, which forms by far the greater division; and part three, which views the subject from the standpoint of laboratory diagnosis.

COLLABORATORS AND CONTRIBUTORS

In the compiling of a supposedly standard, up-to-date hand book of tuberculosis, one especially intended for medical students, I have aimed to include, as nearly as possible, all the various phases of the disorder as they manifest themselves in disturbances in the organs and tissues of the human body; so, in the second part, the section on clinical tuberculosis, I have availed myself in many instances of the services of such of my colleagues who are specialists in the various branches of Medicine and who have made an intensive study of the problems of tuberculosis as they relate to these various branches.

Many of the following chapters are based either upon the lectures delivered or the clinical instruction given before my classes during the various summer quarters at Rush Medical College. Some appear almost in entirety, some have been abstracted, and additions and notations made so as to appear in harmony with the up-to-date teachings concerning tuberculosis of the different organs of the human body.

The credit for this labor and the compiling of these chapters is due to my associates and colleagues as follows:

Chapter 16. "Roentgenology in Pulmonary Tuberculosis." Dr. Cassie Bell Rose, Roentgenologist to the Presbyterian Hospital and Instructor in Surgery (Radiology) Rush Medical College.

Chapter 20. "The Surgical Treatment of Pulmonary Tuberculosis." That part of this chapter which describes the technic for the treatment of pulmonary tuberculosis by means of artificial pneumothorax is based upon a lecture delivered and the clinical demonstration for the induction of lung compression given by Dr. Everett Morris, formerly Medical Superintendent of the Oak Forest, Illinois, Tuberculosis Sanatorium.

Chapter 22. "Physiotherapy—Occupational, Vocational." Dr. Clarence L. Wheaton, Instructor in Medicine.

Chapter 23. "Tuberculosis in Children." That part of the physical examination, "Radiography, or the Roentgenology of Tuberculosis in Children," should also be accredited to the labors of Dr. Cassie Bell Rose, and the treatment of Tuberculous Adenitis be accredited to Dr. A. R. Metz, Surgeon and Roentgenologist at the Washington Boulevard Hospital.

Chapter 26. "Tuberculosis and Pregnancy." Dr. C. Henry Davis, Milwaukee, Wisconsin. Formerly Assistant Professor of Obstetrics and Gynecology at Rush.

Chapter 28. "Tuberculous Laryngitis." Dr. Elmer L. Kenyon, Assistant Professor of Laryngology and Otology.

Chapter 30. "Tuberculosis of Bones and Joints." Dr. Edwin W. Ryerson, Associate Professor of Surgery (Orthopedic).

Chapter 31. "Tuberculosis of the Genito-Urinary Organs." Dr. Herman L. Kretschmer, Assistant Professor of Surgery (Genito-Urinary).

Chapter 32. "Tuberculosis of the Skin." Dr. Edward A. Oliver, Assistant Professor of Skin and Venereal Diseases.

Chapter 33. "Tuberculosis of the Eye." Dr. William G. Reeder, Assistant Professor of Ophthalmology.

Chapter 37. In the third part of this book—"The Blood in Tuberculosis." "Complement-fixation in Tuberculosis"—Dr. H. J. Corper, formerly in charge of the Pathological Laboratory of the Municipal Tuberculosis Sanatorium of this city, has kindly contributed this part of the chapter which is given somewhat in detail.

Others, who in no less degree have contributed to the success of this volume, should here be mentioned—The late Dr. Willard W. Dicker, who for many years was associated with me in tuberculosis work at the Municipal Tuberculosis Dispensary at Rush Medical College. To Drs. Fuller B. Bailey and Rollin H. Moser, internes at the Washington Boulevard Hospital, for their kind contributions to that part of the volume covering the field of physical exploration of the chest, to Dr. Harry Gauss, Denver, Colorado, who so kindly suggested some of the necessary corrections, additions and notations to Chapter 37—"The Blood in Tuberculosis." To the Librarian, Miss Catherine A. McAuliff, and Assistant Librarian, Miss Anna P. McAuliff of the Rush Medical College Library for their kind assistance in looking up the literature on various tuberculosis topics, and to Miss Eleanor P. Fox for the arranging and executing of the necessary stenographic manuscripts, notes, etc.

I am most grateful and under lasting obligation to all for the generous collegiate spirit shown, for the noble contribution of both time and labor, which alone was the incentive which enabled me to present to the student body a concise and clear survey of this most intricate, interesting, and engaging problem—"The Study of Tuberculosis."

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PART ONE

THE PURELY ACADEMIC QUESTIONS IN THE STUDY OF TUBERCULOSIS

In order to be able to understand the various tuberculosis problems arising, we must at the very beginning of this study, at the very threshold, have a clear conception of how these microorganisms first became implanted in the human body and later produced active disorder. Being a germ disease, we ask intuitively whence come these germs, how do they gain access into our bodies, and how do they produce or bring about disturbances? This brings us to an intensive and interesting study of various questions, most of which have only a more or less academic importance. These questions will be taken up separately in orderly sequence, divided into nine chapters.

CHAPTER 1

THE HISTORY OF TUBERCULOSIS

Tuberculosis, the most widely disseminated and most chronic of all diseases, was well known to the ancients and, although no special mention is made in the writings of the Egyptians, Babylonians, Chinese, Jews, etc., all primitive races were undoubtedly acquainted with this disease (13), (30).

Hippocrates, B. C. 464-376, the father of Medicine, has handed down to us a good description of pulmonary as well as laryngeal tuberculosis. His classic description of the clinical nature of tuberculosis had not been reached for more than fifteen hundred years (A. C.) He speaks of fever, hemorrhage, cough, pain in chest and pleurisy, of clubbing of the fingers, recognized the chronicity of the disease, considered tuberculosis contagious, gives most ingenious directions for the arrest of the disease, to remain much out of doors, to drink freely of milk, to avoid intoxicants, venereal and other excesses and advised a change of climate. Aristotle, B. C. 384-322, mentions that the Greeks considered tuberculosis very contagious. Celsus B. C. 30-A. C. 50, although not a physician, recognized the ulcerative processes of both the larynx and trachea; and Pliny, A. C. 79, recommends a sojourn in the pine forests. Galenus or Galen, A. C. 131-201, recognized ulcerative tuberculosis of the larynx and its connection with the lungs. He opined that the ulceration was primary and that the irritating secretions from the ulcer infected the lungs. He recommends the free use of milk and a residence in the mountains. Areteus[1] (A. C. 200) classic description gives us the first clear picture of phthisis. He recognized the difference between tuberculosis and emphyaema, recommended a sea voyage and country air for the cure of the disease; and Avicenna,

[1]It may be of interest to note in studying the history of tuberculosis that in remote times, in antiquity, the observers were chiefly concerned with the symptomatology of tuberculosis, that later in the writings of Sylvius, Fracastori and others of their time, we observe the first clear conception of the tubercle as the fundamental anatomical lesion of the disease. During the 17th and 18th century, the anatomists concerned themselves with the anatomy and the pathology, and during the 19th century, the beginning of the experimental epoch, which led to the discovery of the bacillus as the etiological factor of the disease, the true fundamental conception of tuberculosis in all its protean forms, the exact nature of the disease was established.

A. C. 1000, an Arabian physician, mentions that phthisis is produced by ulceration in the respiratory apparatus, small ulcers healing large ones remaining incurable.

From Galen's time until the 16th century, with the exception of Avicenna, no mention is made of tuberculosis as a disease entity, and in that century the first attempt was made at an anatomical and physiological orientation.

Paracelsus, 1493-1541, wrote a comprehensive treatise on acute and chronic diseases consisting of eight books in which he speaks at length on tuberculosis; Sylvius, 1478-1555, (Latinized from Jacques du Bois) a French anatomist, first recognized the tubercles which he considered to be glands analogous to scrofula and upon which depended the scrofulous inheritance. He believed in the contagiousness of the disease and associated scrofula with tuberculosis, and recognized the existence of lymph nodes in connection with suppuration of the lungs.

Fracastori, 1483-1553, a native of Verona, attributed the origin of tuberculosis to extremely minute germs or seeds, anticipating our present germ theory of disease. He maintained the transmissibility of the disease from the breath of the tuberculous.

Fabricius, 1537-1619, a pupil of Fallopius whom he succeeded as Anatomist at the University of Padua and who was a teacher of Harvey, the supposed discoverer of the circulation of the blood, records a number of dissections, both pulmonary and mesenteric and published a monograph on the anatomy and physiology of the larynx. Forester in his published book at Rouen 1653, described a variety of cases of tuberculosis as observed by him.

Richard Morton's book entitled "Phthisiologia" 1689 says: "Yea, when I consider with myself how often in one year there is cause enough ministered for producing these swellings even to those that are wont to observe the strictest rules of living, I can not sufficiently admire that any one at least after he came to the flower of his youth can die without a touch of consumption." He believed in the heredity and contagion of the disease.

Morgagni, 1688-1771, doubts the identity of tubercles and glands as had been taught, and he would not dissect the body of the phthisical. We notice, here for the first time a fear expressed as to tuberculosis. It is worthy of note that in 1782, just 100 years before the discovery of the tubercule bacillus, a dogma was issued at Naples, of the infallible contagiousness of

tuberculosis and that all bedding, clothing and wearing apparel belonging to the deceased tuberculous must be destroyed or burned.

Benjamin Rush, 1783, in his "Thoughts upon the cause and cure of Pulmonary Tuberculosis" firmly believed it contagious and Matthew Baillie, 1793, in his book on morbid anatomy shows more exact observation, describes the tubercules as firm white bodies dispersed throughout the lungs and which are apparently found in the cellular structure.

Mangetus, 1790, recording his observations states that in a post mortem, he found little bodies which he compared in size and appearance to that of millet seeds. He found these little bodies in the lungs, liver, spleen and other organs and regarded them as phthisical. He believed that they arose from lymph nodes.

Bayle, 1774-1816, teacher of the great Laennec, was the first to attempt a classification of pulmonary tuberculosis on a strictly anatomical basis, a correct teaching, a lesion of the lung causing destruction of tissue. He recognized the disease as occurring in different organs and not limited to the lungs, and concluded that it was not an inflammatory process, but a generalized disease, a diathesis. He classified tuberculosis into six varieties and correctly described miliary tuberculosis.

Laennec, 1781-1826, the father of modern auscultation, taught the unity of phthisis in all of its various forms. He recognized scrofula as tuberculosis of the lymph nodes, recognized its specific character, a pathological entity, which is characterized by the tubercle as an accidental product foreign to the normal healthy tissue. He recognized a favorable soil and a healthy seed. He also recognized the tubercle as undergoing change, softening, caseation and finally breaking down from the center and discharging pus. He distinguished gangrene from carcinoma and established the true theory of unity. He taught that all tuberculosis depends upon the tubercle, that phthisis and tuberculosis are identical, that tuberculosis is an accidental product in the lung, a neoplasm and considered scrofula gland tuberculous.

Broussais, 1772-1838, and his school opposed the teaching of Laennac, believing in the doctrine of chronic inflammation of the lungs, and considered this to be of two kinds, namely, that of the blood vessels and that of the lymph channels. The latter is

tuberculization and is preceded by the first as pneumonia and pleurisy. With the improved microscope at that time he was able to recognize irregular nuclei containing cells which he considered pathognomonic of tuberculosis. Lebert, 1844, came next. He recognized a tuberculous corpuscle with degenerated nuclei and epithelial cells. In 1847 Virchow advanced the dualistic theory. All products of the tubercle are inflammatory and bear directly no relation to the tuberculous process. He recognized only the miliary tubercle. Lymph glands are secondary to an inflammatory area; if glands are enlarged and not inflammatory, it is called scrofula. He recognized the tubercle as a neoplasm, a cellular but not a vascular structure, and that the tubercle may caseate, undergo calcification or fatty degeneration, and is then absorbed and the process healed. The expression prevailed that the greatest misfortune that can befall a phthisical patient is when he becomes tuberculous. The tubercle was a non-specific process, a lymphoma.

Buhl, 1857, states: "Acute miliary tuberculosis depends upon preexisting caseating products which may enter the blood and produce miliary tuberculosis in other organs."

Cruveilhier in 1862 produced artificial tubercles in the lungs, liver, mesentary, etc., by injecting mercury intratracheally and intravascularly, and concluded that tubercles are not specific products, endeavoring to discredit the labors of Virchow. Up to this time the transmissibility of tuberculosis from the dead was firmly believed. Morgagni as well as Valsalva would not dissect a tuberculous subject and Laennec, who died from tuberculosis (after returning from Paris he lived for ten years—during all this time fighting the disease) maintained to the last that he was infected at the dissecting table. Morton, 1834, a student of Laennec, in a treatise on tuberculosis published in Philadelphia, stated that the disease was due to altered secretions and not to inflammation. He believed in the open air treatment of the disease. Although Kortum, 1789, first tried inoculation experiments on himself with negative results, the true experimental epoch began with Klenke in 1843. He first produced artificial tuberculosis in rabbits. He inoculated tuberculous material into the vein of a rabbit which was followed in 26 weeks by active tuberculosis. Klenke never fully realized the importance of this discovery and for nearly twenty years no further advance was made, and it was left to Villemin, 1865, to first demonstrate the

transmissibility of this disease. His discovery caused a furore in the medical world. He inoculated rabbits from man, from cows and from tuberculous rabbits, intravenously. This brought about a complete change in the conception of this disease. He demonstrated first that tuberculosis was a specific disease, second that it possessed an inoculable virus, third that the transmission from man to rabbits and other animals was positive, and fourth, that it was a virulent, dangerous disease, and should be classed with small pox, syphilis, scarlet fever, etc.

Chaveaux, 1868, first demonstrated the transmissibility of Bovine tuberculosis via the alimentary canal. Weigert, 1879, demonstrated the positiveness of miliary tuberculosis to venous infection. Edwin Klebs, 1877, was able to develop on egg albumen an organism which he described as Monas Tuberculosum, a contagion vivum. This now gave new impetus to the histological study of the tubercle, and the discovery of the giant cells followed with their multiple nuclei described by Rokitansky, Virchow, Klebs, etc. Next tubercles were described by Kőster, 1876, in joints, by Schueppel in lymph glands, by Friedlander, 1873, in Lupus, by Lannelongue in cold abscess. From these numerous observations and deductions it was most evident that the true nature of the tuberculosis virus, the etiologic factor of the disease, would be within human possibility of demonstration, and much scientific experimental work was undertaken by pathologists and bacteriologists all over the civilized world, but it remained to Robert Koch, 1882, and again in 1884, to demonstrate positively the tubercle bacillus as the cause of the tuberculous disease. His memorable paper read March 24, 1882, before the Physiological Society of Berlin was received with great acclaim. His was the most important contribution to the tuberculosis questions. He proved the presence of the tubercle in different pathological structures of the body to be anatomically the same. He proved the unity of tuberculosis, as first taught by Laennec, that tuberculosis of bones, joints, glands lungs and all other organs of the body is produced by one and the same organism, the tubercle bacillus, immaterial however different the anatomical characteristics; and when again in 1890 he was able to demonstrate the active substance given off by the germ in its growth and which is now generally known as Tuberculin, the medical profession hoped for a realization of that fond vision that at last a cure for tuberculosis had been discovered.

The fiasco and disappointment is still fresh in the minds of many medical men. With the discovery of the tubercle bacillus a most intensive study of tuberculosis began, and the names of Cornet, 1901, Human and Bovine tuberculosis, v. Behring's Cassel Lecture, 1903. Calmette, v. Pirquet, Mantoux, Marcus Paterson, Nathan Raw, Edward L. Trudeau, Theobold Smith and a host of others will always appear as shining lights in the study of the history of tuberculosis.

CHAPTER 2

THE ETIOLOGY OF TUBERCULOSIS

THE TUBERCLE BACILLUS

Syn.: Koch's bacillus, Phthisis bacillus, etc.

If Klenke in 1843 anticipated the etiology of tuberculosis and Villemin in 1865 definitely proved that the virus as found in tuberculous tissue or material was transmissable to both man and animals, it was left to the master mind of Robert Koch to demonstrate in 1882 the causative factor, the tubercle bacillus, as the active virus of the disease. This bacillus, so called from its rod shaped appearance, the etiological factor of all tuberculous disease, is a small fungus, a mushroom, a single celled plant and as such it is governed by the same definite laws that governs all plant and animal life, that is, it grows, multiplies, propagates the species and dies, and throughout all its growth it is governed also by the same laws of metabolism that obtains in all plants and animals. As the human being eliminates during its existence various toxic bodies, urea from the ingested proteins, carbonic acid gas from the inhaled air, so does the tubercle bacillus in its growth give off a toxic product. This product is known as tuberculin.

The tubercle bacillus has never been found to exist free in nature. We know not its origin and neither do we know if it has always existed in its present or in a different form or if it has assumed a different shape or form in the human or animal body. From our present knowledge concerning the bacillus, we know it to be an obligative parasite requiring for its sustenance and subsistence, animal and body heat and nutritious material. Outside of a host it can not thrive.

Types and Varieties

From the cultural growth and the pathogenicity of the (50) tubercle bacillus we now recognize about four distinct types, all producing tuberculous disease in man or animals; namely: (a) The Bacillus Tuberculosis Humanus (Homo). Found chiefly in the human.

(b) The Bacillus Tuberculosis Bovinus (bos). Found in cattle and occasionally in man.
As both human and the bovine bacilli are concerned in the tuberculous processes as observed in man the various characteristics of these two types will be given more in detail throughout this chapter.

(c) The Bacillus Tuberculosis Avium (avis). (Producing Tuberculosis Gallinarum or Fowl Tuberculosis.) This bacillus is found chiefly in fowl, like chickens, birds, doves, however, ducks and geese are comparatively free. Morphologically it simulates closely the human and is not pathogenic to either man or cattle.

(d) The Bacillus Tuberculosis Piscium (Piscis).
The bacillus of cold blooded animals (Poikilothermal) found in fish, reptiles, turtles, and occasionally in worms described as the blind-schleichen (a species of worm). This bacillus also resembles morphologically the human is, however, avirulent to guinea pigs, rabbits and doves, virulent to fish, to reptiles and all amphibia; frogs are very susceptible. The possibility of successfully inoculating cold blooded animals with bacilli of the bovine or avian type is still a question in dispute.

Other Varieties

(e) The Bacillus Leprae. The leprosy bacillus also belongs to this, the acid fast group. This bacillus morphologically resembles very much the human type. It is found, however, in the diseased tissue in dense masses and intracellular whereas the tubercle bacillus is found in small groups and extracellular. As a rule animals are not sensitive to this bacillus.

(f) The Bacillus Smegmatis. This bacillus resembles the human type, is acid fast but is less alcohol fast and is not pathogenic to test animals. This bacillus is frequently found about the genital organs of both male and female at any age and may then become contaminated with the urine and if so may be mistaken for the true tubercle bacillus. Here animal inoculation will give the differential test. This bacillus at times is also found in ear wax, nasal secretions, etc.

(g) The Bacillus Pseudotuberculosis or Pseudobacillus (also Bacillus tuberculoides). This bacillus is found very much diffused in nature, may be one and the same organism, saprophitic and acid fast, resembling the different varieties mentioned. It is variously described as milk, butter, manure, bowel content, grass, timothy grass No. I and timothy grass No. II, trumpet, earth bacillus, etc.

Both the Pseudo and the Smegma bacillus are non-pathogenic.

It is now generally recognized that a close relationship exists between the human and the bovine bacillus. Perhaps both (or may be all) have been derived from a common ancestor but whether they have become specialized or modified by adaptation to the different host can, for the present, with our knowledge concerning these organisms, not be definitely answered, hence, the question of the identity or nonidentity of human or bovine strains has not been definitely decided. Three views, however, are generally held, (1) that the human and the bovine are distinctly different, (2) that both forms are identical, are only varieties of one and the same kind and (3) that they are different in kind but transitional in forms.

Description and Characteristics of the Tubercle Bacillus
(From L. Bacillus or Bacculus a stick or staff)
Morphology. Pleomorphism.

The tubercle bacillus is a distinct, definite and slow growing organism about 2.0 to 4.0 microns (millionth part of a meter), 1/8000 of an inch in length, about 0.3 microns thick (1/100000 inch) equal to half the diameter of a red blood corpuscle and about 1/8 to 1/10 as wide as it is long. (59). As we observe it, when stained and under the microscope, it is usually slightly curved, the ends somewhat rounded. When cultivated under various conditions, it grows with more or less vigor assuming various shapes. This is referred to as pleomorphism, and when grown under the most favorable conditions, branching threads are given off or clubbing may be observed at one or both ends, and in old lung cavities the bacillus is often found growing in long chains. The bacillus is a parasite midway between bacteria and streptothrices, is non-motile, and owing to its pleomorphism is considered undergoing a cycle to a higher development, a

tuberculomyces related to and approaching the ray fungus, actinomyces. The various types with which we are familiar, namely, the human, bovine, avian, reptilian, are seemingly all interrelated. The bacillus when growing does not produce spores and vacuoles which have been occasionally observed are supposed to be due to degenerative changes. As to its viability, it is very little affected by cold, is very resistant to external influences, dies at a moist temperature of 80°C. or 175°F., or a dry heat of 100°C.=212°F. Direct sunlight destroys it within a few minutes to half an hour and diffuse day light in from half an hour to a day. A 95% alcohol kills the bacillus in from one to fifteen minutes, it dies quickly in a 5% carbolic acid, or in a 1/10 of 1% mercuric chloride solution. In the dried sputum the bacillus may lose its virulence in about three months but under favorable condition may retain it for more than eight.

Tinctorial Characteristics. The bacilli of this class all belong to what is known as the acid fast group, that is, when stained with aniline dyes and subjected to alcohol and acid treatment they do not give up the color. They have great affinity or predilection for red dyes, particularly fuchsin, which when once stained they hold with great tenacity. This peculiarity is said to be due to the presence of a fatty acid in the body envelope. (Waxy body envelope is disputed by many.)

Cultural Characteristics. By far the most interesting peculiarities of the tubercle bacillus are found in its growth and by means of this we are able to differentiate the various types. The human bacillus is strictly aerobic, requiring air for its growth, whilst the bovine is anaerobic requiring little or no air. The human bacillus is somewhat longer, more slender and slightly curved, whereas the bovine is broader, slightly thicker and more plump.

On blood serum (human) the human cultures grow vigorously often profusely and multiply freely while bovine colonies grow much more slowly and very irregularly. A vigorous growth of the bovine type, on a neutral 5% glycerine bullion will convert such a media into an alkaline substance, not so the human. If at the end of a month's growth of the bovine type on such a solution an alkaline reaction is given, the growth of the human on a similar solution will give an acid reaction and the degree of acidity may reach as high as 4%, hence we notice that the

human grows more freely in an acid and the bovine as well as the avian best in an alkaline media. (Prof. Theobald Smith.) On a potato media with bovine bile the bovine bacillus grows very vigorously and both the human and avian very sparingly, whilst on a potato media with human bile, the human bacillus grows luxuriantly and the bovine and the avian poorly, on a similar media with avian bile all show a very vigorous growth.

Pathogenic Characteristics. The pathogenicity of the different types, that is the human and the bovine, is strikingly and peculiarly different. We find the bovine bacillus to be highly toxic to rabbits and cattle, retaining this virulence even after prolonged cultivation, with great tenacity for these test animals. Not so the human, but the human is very virulent to guinea pigs, slightly to rabbits and seldom to cattle. In man the bovine bacillus may be found in glands, tonsils, in the intestines, but is usually not virulent and serious disease occurs but rarely. The human bacillus is seldom found in the carnivora, in birds, in fowl, in reptiles, the bovine very frequently. It is impossible from the lesion alone to differentiate the human from the bovine and vice versa, nor can we morphologically distinguish between human and bovine tuberculosis. The bovine bacillus is much more virulent to test animals than is the human, and bacilli are much more virulent if taken from their own source, the human from man, the bovine from cattle.

Infecting Characteristics. It has repeatedly been observed that both the human and the bovine bacillus have a special predilection for various organs and tissue of the human body, and that while the human bacillus selects such organs as the lungs, the pleura, the larynx, we find that the bovine bacillus usually spares these organs and favors the glands, the bones, the joints, the peritoneum, the skin, etc.

In man pulmonary tuberculosis induced by the bovine bacillus is extremely infrequent. The English commission after 10 years of careful observation reports that in about 6/10 of 1% of all cases of pulmonary tuberculosis the bovine bacillus is the etiological factor. According to Koch human beings may become infected with bovine bacilli but infrequently become tuberculously diseased. Human bacilli have not been found in cattle; bovine bacilli have been found in man in glands and in the intestinal tract, but they usually show no virulence and remain localized.

Chemical Compositions of the bacillus. On account of its chemical composition, richness in fat and wax the bacillus possesses special resistance to the ordinary stains or aniline dyes, but if once stained it shows an exceptional tenacity or resistance to decolorization or destaining.

The tubercle bacillus can be dissociated into its component parts by various methods. This is mainly affected by the use of diluted or weak organic acids such as lactic, malic, phosphoric, etc. Careful observations and analysis have demonstrated the bacillus to consist of 85% of a watery substance and 15% of a dried residue, and of this residue approximately 25% consists of a fatty substance soluble in alcohol and ether. Of this fat about ¼ or 25% consist of free fatty acid and 75% of neutral fat or wax. Of the other constituents, a protein, (toxic) chiefly nucleo-albumin is the most abundant, and cellulose mineral substances, potassium, sodium and calcium oxides and some carbo-hydrate supply the remaining constituents. On incineration about 8% of ash remains. A small percentage of an odoriferous principle is also present.

Special Adaptation Characteristics. Bovine bacilli are different from the human and there are atypical types which strictly speaking belong neither to the human nor to the bovine; these types may be simply varieties of one and the same organism or intermediate types. The organisms evince also a special adaptation to different hosts, for we know that the strains of the human kind may adapt themselves to the bovine organism, become virulent and assume the cultural characteristics of the bovine, and yet it is questioned if the bovine bacillus infecting man ever assumes the characteristics of the human. The time has not yet arrived when we can say positively that a transformability has taken place or that one strain has been converted into the other, the human into the bovine and vice versa; the increase or decrease of virulence by passage through the body proves nothing. There are many aberrant types of more or less variation in their growths. Prof. Theobald Smith removed from the neck glands of a child bacilli which showed the characteristics of the bovine type but also showed an unusually slow growth, retaining this same peculiarity after 6½ years' cultivation. Animal transmissions vary greatly. As a rule swine do not transmit to others of the same species. Again, the passage of the human type

through an animal, as for instance, the goat, may greatly increase its virulence.*†

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*The tubercle bacillus is as stated a minute vegetable organism, the human, bovine and all others. There is no tuberculosis without the presence of this bacillus, no matter how susceptible the being is. Everybody is susceptible to the action of the bacilli after they have entered the body, but not everybody becomes tuberculously diseased. There are always three factors: bacillus soil and opportunity. In some the infection causes only a slight disturbance (30%); in some the disease becomes manifest or active (10%); and the remaining 60% through life are not aware of the presence of the bacillus in the body.

†In recent years Dr. Jaimé Ferrán (89), Catalonia, attracted considerable attention, giving his views based on both clinical and laboratory observations as to the infectiousness of the tuberculosis virus. He maintains that the tubercle bacillus in pure culture if inoculated into man or animal, is incapable of producing acute tuberculous disease such as is recognized by beginning granulation tissue or early death, but that if tuberculous sputum or tuberculous tissue is used as inoculating material, it will produce typical tuberculosis even if the tubercle bacillus can not be demonstrated to be present. In this expectorated material or in the tissue various non-acid fast bacteria are present which are capable of bringing about an acute or chronic form of tuberculosis. From the many different bacteria present in a tuberculous process, the acid fast bacillus of Koch is only one of the many varieties derived from an ordinary non-acid fast saphrophite which can be cultivated and which differs materially in its tinctorial characteristic from the ordinary tubercle bacillus.

Much's Granules (89). The tubercle bacillus in granular form. It has been proved that occasionally in tuberculous material a virus is present which will not take the ordinary Ziehl-Neelsen stain but can only be stained with a modified Gram. These bacillary granules, remnants or salvage, are a form of tubercle bacilli which are not acid fast, they are, however, the normal parts of the bacillary bodies. Moreover, they represent the clinically most resistant parts of the tubercle bacillus, the parts which survived after the disintegration of their bodies, is usually a virulent form and a peculiar developmental stage. These granules found in tuberculous material are an expression of a positive reaction of the defense mechanism of the body. The rod shaped bacteria of less resistance or virulence have been destroyed by this mechanism of defense and these granular bodies are simply the remains of this destruction, a subsequent lowering of the body's resistance may again change these granules into the rod shaped form. If these granules are placed into the body of a susceptable animal they are again transformed into rod shaped bodies. That these granules do not take the ordinary Carbol-Fuchsin stain, but take up readily a modified Gram is due to the fact that the fatty portion or envelope of the tubercle bacillus is absent—is wanting in the granular form. It is this fatty component of the bacillary bodies which gives the acid fast character. Possibly the non-acid fast bacteria occasionally found in tuberculous material and to which Ferrán alludes may after all be nothing more than Much's granular form of the tubercle bacillus.

CHAPTER 3

THE PORTALS OR ATRIA OF INFECTION

PORTA INFECTIONIS

Query: In what way is the human body infected? How does the germ gain entrance into the human body? How is tuberculous infection brought about? What are the possibilities of infection? In short, what are the avenues, atria or portals of infection?[1]

There are two recognized avenues of infection, an external or cutaneous one, that is by way of the skin, and an internal one through the mucous membrane. The human body consists of a firm bony framework to which are attached the various muscles, and interspersed between and throughout them are the internal organs, the blood vessels, glands, tissues, etc., all encased in a double sack of which the Lamina Externa or skin forms the outer investment, and the mucous membrane, the inner, the Lamina Interna. If both layers of this sack, the cutaneous and the mucous, are perfectly intact, then, relatively speaking, all the various bacteria both pathogenic and non-pathogenic will be incapable of entering the body, and hence will be incapable of producing disturbances. Infection through the skin is extremely infrequent and then only after more or less denudation and a healthy mucous membrane is also, relatively speaking, capable of withstanding the entrance of bacteria. It is only when by means of irritants the mucous surfaces become congested, or the invaders are in too great a number that entrance may be gained

The atria of infection, the primary portals, as usually observed may conveniently be subdivided into five routes, namely:

1. The aerogenous, the bronchogenic route, that is by inhalation through the respired air;

2. The enterogenous, the intestial route, that is by ingestion, by deglutition;

[1]The entrance of the tubercle bacillus into the human organism may result disastrously or may be perfectly innocuous depending upon many factors, namely, (1) difference in the degree of susceptibility in different individuals; (2) difference in the degree of virulence of the bacillus; (3) the number and the amount of bacilli entering the body at a given time; (4) if the infection was single or if it was oft repeated; (5) the age of the individual if healthy or not at the time of infecton, previous diseases, sanitary and hygienic conditions, etc. This latter is usually an acquired susceptibility or disposition; (6) the points of entrance, if through the skin, tonsils, mouth, intestines, etc.

3. The cutaneous route, through the skin;

4. The genitogenetic route, congenital and inherited, including spermatogenous, ovagenous, germogenous, placental, etc.;

5. The crytogenetic route, that is an obscure or unknown route.

The lymphogenous and hematogenous infection so frequently spoken of are secondary processes of infection and not of this class.

1.—**The Aerogenous Route.** The chief portals of infection are through the various mucous surfaces of the upper air passages, those of the nose, mouth, pharynx, larynx, as well as those of the bronchi. The vibrissae about the nasal passages mechanically filter the respired air and in this manner at least 95% of all dust and other particles are removed. Besides this, the mucous secretions possess bacteria destroying properties. The glands of the Waldeyer ring, tonsils, adenoids, lingual tonsil, etc., guarding the entrance to the lower air passages if healthy offer a barrier to the bacilli, but if diseased favor their entry and in this way bacilli may enter the cervical glands. The same holds true of all the mucous surfaces of the oro-pharynx.

After careful and prolonged observations by competent workers, the view is now entertained that most infections, or at least 85% are by inhalation of air through the upper air passages. There are two theories as to this aerogenous infection. Fluegge maintains that most infection takes place by immediate and intimate contact with the active tuberculous, and that in coughing, sneezing, hawking and spitting, small droplets of sputum containing tubercle bacilli in greater or less number are expectorated, and these droplets floating in the air are inhaled by those who are in close contact with the diseased individual. This method of infection with moist sputum is now generally accepted. Cornet (22) on the other hand emphasizes that it is the dried sputum of the tuberculous which is the chief source, that the tuberculous individual, being frequently a very careless person, coughs and spits about the house, upon the floor and walks, the sputum is tracked about and dried, currents of air carrying the dried particles in all direction and contaminating the respiratory air. In this way the bacilli enter into and find lodgment in the human body. It must be conceded that infection may frequently take place in that way and that infants in particular are infected in that manner. As to which of these two theories is correct can not be stated; most likely both are in part correct.

That infection takes place most frequently by the aerogenous route has been conclusively demonstrated first by Küss and later by Ghon (15), who out of 184 carefully conducted autopsies could prove that in all but one, infection took place through the inhaled air. It is generally conceded that the respired air, through the mouth or nose is, at all times, more or less laden with dust particles and various micro-organisms, both pathogenic, and non-pathogenic, which are conveyed into the bronchial tubes and their ramifications constantly bathing the lungs with air containing many bacteria which thus reach the mucous surfaces where many are deposited. The greater number, however, due to the anatomical structure of the lining membrane of the bronchial tubes, the ciliated epithelial cells, are again expelled along with the bronchial secretions. Yet some will find suitable lodgment, produce irritation causing infection and ultimate involvement of the regional lymph nodes. Phagocytosis here undoubtedly also plays an important role. As has been stated above, in man the aerogenous route is the most frequent and the chief cause of almost all pulmonary tuberculosis as well as that of many forms of metastatic disease, all depending upon the virulence of the organisms and the amount of virus reaching the lungs, that is upon the quality and quantity of the invading bacilli, and also upon the sensitiveness of the host; hence, primarily, tuberculosis is both a quantity and a quality infection.

2.—**The Enterogenous Route.** By ingestion, by deglutition. The alimentary canal is a less frequent atrium or source of tuberculous infection than the aerogenous route. v. Behring (48) believes that the enterogenous atrium is the main source of infection and that by means of infected milk, meats, and other food stuffs infection is brought about in man by way of the digestive tract. It is not at all improbable that with the taking of infected foods through the mouth, bacilli may find entrance into the respiratory tract, but that must be extremely seldom and the food would have to be laden with bacteria. The hypothesis of v. Behring that pulmonary tuberculosis in children takes place by the aspiration of tuberculous material through the stomach and intestines is improbable. It is more reasonable to assume that with the ingestion of food particles containing tuberculous material, bacilli may find entrance into the respiratory tract and here again, as has already been stated, the food would have to be saturated with the virus.

The number of cases of primary intestinal infection must always be relatively small, but we may assume that if tubercle bacilli enter the intestinal tract in large numbers, or the mucous membrane be irritated or not intact, that then infection may take place. It has never been proven satisfactorily that the ingestion of cow's milk containing bovine bacilli has caused pulmonary tuberculosis in man, and concerning the meat of a tuberculous animal as a rule it does not contain living tubercle bacilli. For this to take place the animal must be generally infected but then the meat would not be saleable and even in that event the thorough boiling would kill the bacilli. In the alimentary tract many bacilli are killed and few if any pass into the mesenteric or retroperitoneal glands. It has never been proven that tubercle bacilli pass the normal mucous membrane of the intestines and infect the regional glands. A small infecting material and an intact intestinal canal will always inhibit infection and only if the bacilli enter the alimentary canal in large numbers or if the mucous surfaces be not intact will infections take place. Even infants are very infrequently infected from the milk of the tuberculous mother, but this may take place when the glands of the mammae are themselves tuberculous. In about 10 to 12% the infection in the human is due to the bovine bacillus and then it is usually found in children manifested in gland, bone and joint tuberculosis, in about 85%, mainly in adults, it is due to the human bacillus. In the human, the infection takes place from man to man, that is from a tuberculous adult to an infant, and only a small percentage of children (one-eighth of all) tuberculously infected show evidence of an entrogenous infection. Intestinal tuberculosis in the adult is usually always secondary to pulmonary disease, but may be primary in children from milk containing bacilli. The oesophagus and stomach play no part in the way of infecting atria and we find that these organs are most infrequently primarily or even secondarily tuberculously diseased, however, in the active tuberculous a secondary process may follow an injury to these organs. It may be stated that buccal, nasal, laryngeal or pharyngeal primary tuberculosis is also extremely rare. It is most difficult to prove that a tuberculously infected tonsil or an adenoid vegetation, as a primary infectious atria, could later be followed by a pulmonary lesion, although in manifest tuberculosis we find a very frequent tonsillar infection, but this is secondary. It may be worthy of note here that in

pulmonary tuberculosis the cervical lymph nodes are very infrequently simultaneously involved.

3.—**The Cutaneous or Dermal Route, Through the Skin.** The next most important route of infection. As stated above, if the skin is intact, bacilli can not enter the human body—an abrasion is always necessary to bring about infection. Abrasions are usually noticeable about the nose, mouth, eyes and anus, that is, where the cutaneous and mucous surfaces come together, and infection may also take place through other parts of the external body, the skin of the arms, hands, etc., if the cuticle is not intact. Mention may be made of the condition known as Tuberculosis Verrucosa Cutis or Tuberculous Warts which may be observed on the hands of undertakers, embalmers, tanners, butchers, cattle dealers and those who handle dead bodies. Other cases of infection through the skin are recorded where in vaccination the vaccine had been contaminated with the saliva of the tuberculous vaccinator, transferring in this way the tubercle bacillus directly into the body of the person being vaccinated. In medical literature (89), mention is made of tuberculosis following circumcision in which the infection was brought about by bringing the sputum of the tuberculous individual performing the rite in direct contact with the wound. Very frequently in years gone by has tuberculosis been innocently transmitted by an irresponsible tattooer who carelessly and with indifference moistened the india ink being used with his bacilli laden sputum, perhaps not being aware that he was tuberculously diseased. Tuberculosis has also been transmitted in coition and cases of tuberculosis following trauma by direct infection of the wound, primarily, have repeatedly been proven.

In a pathological laboratory some years ago a young physician while handling a flask containing live tubercle bacilli had the misfortune to break it and pour its contents over his bare arm. Though the arm was immediately washed and the skin sterilized, the incident was followed later by active tuberculosis. It remains still a disputed question whether living tubercle bacilli being present in large numbers, actually penetrated the intact cutaneous surface. Is it not more probable that the incident of breaking the flask brought about mental disturbances which caused a lowering of body resistance and consequent disease?

4.—**The Genitogenetic Route, Congenital or Inherited.** Placental transmission and transmission via the maternal ova produce true congenital tuberculosis and are the only true sources

of maternal hereditary infection. Congenital tuberculosis may have various atria; conceptional, germinal, spermatic, ovarian, placental, vaginal, uterine, etc. The spermatogenous or ovagenous route as a primary way of infection must be extremely rare. In fact, no direct proofs have yet been submitted as to the certainty of infection by this means. Primary tuberculosis of the female genital organs, the vagina and uterus, are also infrequent, there being no direct proof of this mode of infection.

Placental Transmission. Transmission may take place through a diseased placenta. In order to prove the congenital transmission of the tuberculosis virus, it is not sufficient to demonstrate the tubercle bacilli in the placenta; the proof must show that bacilli have actually passed through the placenta into the organs of the foetus, and a negative histological examination of the foetus is insufficient to exclude placental transmission; antiformin methods and animal tests may be necessary. Then again, a tuberculous infection or transmission may take place in embryonal or foetal life through an apparently normal or perfectly healthy placenta. There are now two theories of this early or infantile infection. (1) The intrauterine, congenital or prenatal, which presupposes that all infection takes place before birth, producing a true inherited tuberculosis from the father like syphilis, or a congenital or hereditary infection from the mother; (2) Extrauterine, postgenital or postnatal, which explains that nearly all infection takes place after birth from a tuberculous adult in intimate contact with the infant immediately after birth. v. Baumgarten maintains that many more than we suppose are infected intrauterinely and yet the anatomic-pathological lesions are exceptionally few before birth, being greater after birth. It is very theoretical to suppose that tubercle bacilli have been transmitted into the young organism by means of the sperm or the ova, or through the placenta without producing any disease, and that they remain dormant in the child until puberty or probably years thereafter, and only then attack the host. Particularly is this true when we consider that the specific tuberculin test in the very young, in infant life, is usually negative, and that it only becomes positive with advancing years, and that infants born with manifest disease all succumb to active generalized tuberculosis in the first months or within the first year of life. From this we must conclude what is now maintained by the greater number of tuberculosis workers, namely, that most in-

fection is extrauterine or postnatal and is brought about after the birth of the child, being only very exceptional before birth.

5.—**The Cryptogenetic Route.** In a very small percentage of tuberculously infected infants and children at autopsy or otherwise (in about half of 1%), it is impossible by the most painstaking procedure to demonstrate definitely in what manner infection has taken place. These cases are now usually classified under the heading of obscure or indefinite infection, or more properly as cryptogenetic.

In this connection it may be of interest to note that v. Behring and his school maintain that the chief source of infection in the human is by the enterogenous route, this through the ingestion of milk from tuberculously infected cattle. However, the consensus of opinion of most competent observers is that tuberculosis is an infection from man to man and more recent studies seem to confirm this view.

A. Balfour states that the great frequency of tuberculosis in the Sudan is due to the increased number of the European people, that there exists relatively infrequent tuberculosis in cattle, but that dogs and apes are very susceptible.

A. Calmette says that in the French colonies in Africa (89), Senegal gold coast, Madagascar, Algeria, tuberculosis of the black races, of the natives, is extremely infrequent, but the disease increases with increased arrivals of Europeans. These races possess no inherited immunity but succumb very readily to the infection. The frequency of intestinal tuberculosis, as is advocated by v. Behring said to be due to infected milk. cannot here be accepted.

H. Zieman finds that tuberculosis is very little disseminated in the German colonies in Africa at Kamerum. In many peoples it is not known and there is no tuberculosis in animals. Tribes exist in the interior of Africa which are still free from tuberculosis and that cattle are also free. The black races fall very easy prey to catarrhal diseases of the respiratory air passages; hence, to tuberculous disease. With increase of trade, social conditions and constant contact with the Europeans the disease increases most rapidly, because they possess no resistance. Chronic bone and joint tuberculosis and lupus are extremely seldom. The Arabians also fall easy prey and according to E. Smith similar conditions exist in Egypt. In Sicily, tuberculosis is on the increase and this is due to the returned Sicilians from the United States. In Turkey, tuberculosis runs a different course from that of the more cultured races. There tuberculosis is very infrequent but also very fatal, less so to the inhabitants of the larger cities. It is very fatal among medical students and military recruits from the country districts. In Palestine, the disease was introduced some years ago by Jews returning from Russia. In Japan, the disease has existed for many ages. In Scotland, tuberculosis of the bones and joints is very prevalent, due to the ingestion of uncooked milk, a pernicious practice which is much in vogue there. In the tropics, in the West Indies, the death rate is from five per thousand to five per ten thousand.

In Australia one per thousand. Bovine tuberculosis and tuberculosis of young children is rare in the tropics. Tuberculous meat is uncommon, milk is always boiled but very little is used. Natives who never came in contact with tuberculosis material become very easy prey to the infection. The natives of India living in cities are less susceptible but the inhabitants of the coast of Africa in the cities are the spreaders of the disease. There also tuberculosis in animals is very infrequent, with the exception of fowl. Hyman reports that in Japan, Greenland, Turkey in Asia, Roumania, Egypt, the gold coast of Africa, the infection is always from man to man. Tuberculosis in South Africa—the British Medical Journal, April 25, 1908—Tuberculosis in South Africa is rampant among the Kaffirs, whereas tuberculosis among cattle is unknown. McVicker, British Journal of Tuberculosis, April 1, 1909, speaking of tuberculosis in South Africa states: "The colored races in western South Africa suffer most severely, both those living in the towns and in the country. The disease is nearly as common amongst the Banta population, in Natal and the natives of the eastern Cape Colonies as well. It is also common among the India settlers in Natal and the Kaffirs, but few cattle are infected and abdominal tuberculosis is uncommon even amongst the Bantas, who are a milk drinking race." In Singapore there exists a higher proportion of tuberculosis among the Chinese male than female—males 6.04, females 3.81. In the Malay peninsula bovine tuberculosis does not exist, no tuberculosis among the domesticated animals. In the abattoirs at Ipoh during the last four years (from 1915 to 1919) more than 250,000 pigs have been killed and no case of tuberculosis has been found showing that the bovine is free from the disease. As a rule Chinese, both adults and children, use but little milk and yet the population of this race who are suffering from tuberculous disease is quite considerable, nearly 5,000 deaths per 1,000,000 population in 1916.

Animal Experimentation. Cattle, as a rule, with advanced pulmonary tuberculosis do not contaminate the milk with tubercle bacilli. It is only when the udder itself is diseased that the milk becomes contaminated. Raw milk may contain in a cc one thousand to fifty thousand or more tubercle bacilli and butter may contain as many as one hundred in each gram. In the feces of cattle suffering from pulmonary tuberculosis, in about 46% it is possible to demonstrate the presence of the tubercle bacillus, even here microscopic examinations are not free from error because many saprophitic acid fast rods are present. Animal test only is reliable, and in the circulating blood of even the advanced cases, tubercle bacilli as a rule cannot be demonstrated. Bang states that large herds of cattle are rarely free from tuberculosis, that all tuberculous animals do not react to the tuberculin test and that in herds in which tuberculosis has existed for years, testing may be omitted because most full grown animals will react although they may appear perfectly healthy.

CHAPTER 4

HEREDITY, DISPOSITION, PREDISPOSITION AND DIATHESIS. THE TUBERCULOUS TENDENCY

Before the etiology of tuberculous disease was well known, much was said and written about the diathesis, the disposition, predisposition and heredity, and even with a better knowledge concerning this disease since the discovery of the tubercle bacillus, there exists still a wide difference of opinion relative to these terms. These terms predisposition, heredity, diathesis or disposition are all more or less vague, indefinite and misleading. What they really mean to convey is that the human body in many instances, especially in infant and young life is incapable of withtanding the implantation of the tubercle bacillus, is the possessor of a fertile soil, that is, in the individual there is a strong tendency toward the disease, hence all these terms are better expressed by the simple words, "The Tendency." This tendency to tuberculosis, first to the implantation of the bacillus and later to tuberculous disease is said to be either an inherited or an acquired disposition; hence, we recognize an (89) idiopathic and a toxipathic disposition or tendency.

First: The **idiopathic,** the **inherited,** the **unavoidable,** the **specific disposition.** The tendency to **tuberculous infection. The** direct (primary) contributory cause of tuberculosis. This presupposes a receptive condition of the body, a heightened tuberculous disposition to infection. This tuberculous tendency or disposition is usually in early life in infants and in small children. There seems to be in infant life a heightened disposition or an hereditary predisposition to tuberculous infection and in later life to tuberculous disease. When a number of infants are exposed under identical tuberculous surroundings or environments, some will develop manifest disease before the expiration of the infant years whilst others may or may not develop the disease after puberty. Because the infant can not protect itself against the infection it is said to be unavoidable and of the former we would then speak as a heightened disposition or a

predisposition and of the latter that the tendency or disposition has lessened.

Second: The **toxipathic**, the **acquired**, the **avoidable**, the **non-specific disposition**. The tendency to tuberculous disease. This is the indirect (secondary) contributing cause of tuberculosis. The extremely great tendency of the tuberculously infected individual to become tuberculously diseased is the tuberculous predisposition to the disease. This predisposition is chiefly found in adult life—the causative factors being many. The idiopathic is prone to a tuberculous infection and the toxipathic to a tuberculous disease, hence in the adult a disposition is really the conversion of a tuberculous infection into a tuberculous disease. In most individuals the infection remains as it is throughout life, does not go further and this is due to the body resistance offered, but in many it does not terminate here, but after the infection it may develop more or less rapidly to a tuberculous disease, and this is really the disposition, the predisposition or diathesis. Strictly speaking there is no predisposition to infection because every human being is prone to infection, but this infection in the greater number of instances does not predispose to disease. A disposition and a diathesis are not one and the same. A diathesis is a form of disposition but a heightened one, an increased tendency to both infection and to disease, and then again we may say that there is no inherited disposition. The infant born of a tuberculous mother inherits only a constitutional weakness which is an anomaly, a lessened resistance of the body to infection, a weakness to a proper functioning of the various organs and tissues of the body, hence a body or tissue weakness. An idiopathic disposition is of prime importance to the implantation of the tubercle bacillus whereas a toxipathic disposition is of prime importance in the development of the disease, for the entry of tubercle bacilli into the organism is not sufficient to produce tuberculosis. The essential conditions necessary are a favorable soil, a virulent bacillus and a receptive organism. Everybody without exception who receives tubercle bacilli below his cuticle, that is to say anywhere into his body, will react to the formation of tuberculous tissue, but it is not necessary that this must inevitably lead to the formation of tuberculous disease. Many heal and again, many remain latent. This is the rule in by far the greater number of cases, but not an absolute rule, because, owing to fact that living bacilli are harbored in the organism in

a measure, an immunity against reinfection from without but not from within is offered. In a mild degree immunity is already acquired, or better, inherited by most infants at birth because nearly all parents, especially of the white race, have at some time in their lives come in contact with the tuberculosis virus and this slight immunity in infant life in a measure already gives to the early infection a definite phase of beginning latency.

The important factors to the question of the idiopathic disposition are the inherited body weaknesses and the inherited exhaustion of the body's power to be able to produce immune or antibodies. Every person possesses in a more or less degree a tendency to a tuberculous infection, evidenced by the fact that at the age of 14 nearly all children are more or less infected, but the tendency or disposition to tuberculous disease declines with advancing years. Take 100 children who are tuberculously infected in the first years of life, nearly all are made tuberculously sick and die; however, this tendency or disposition decreases rapidly with advancing years so that at the age of 6 or 7, only about 20%, and at the age of 14 only about 2% show manifest tuberculous disease, all the other remaining free. After the 14th year of life, towards puberty and thereafter, the acquired, the avoidable disposition or tendency plays the leading part, after this age the toxipathic factors are in the lead as the indirect and contributing causes of active pulmonary tuberculous disease. The toxipathic factors may here be mentioned in the following order of their importance:

1.—**Bad Housing Conditions,** including poorly ventilated living quarters, bad air, insufficient clothing, unwholesome food, want of cleanliness, the underfed, the undernourished, insufficient hygiene of the mouth, teeth, body, etc.

2.—**Mental Disturbances**, such as grief, anxiety, worry, homesickness, fatigue, overwork, overstudy, long hours with insufficient rest, and the various psychic disturbances.

3.—**Toxic Influence Proper.** Alcoholic excesses, inordinate use of tobacco, venereal disease, syphilis, gonorrhoea, etc.

4.—**Mechanical Causes**, like ocupational diseases, inhalation of air laden with dust, coal dust, etc., inhalation of obnoxious gases and vapors, injuries to the chest wall, etc.

5.—**Infectious diseases.** It is well known that after measles, scarlet fever, whooping cough, typhoid fever, pneumonia, etc., pulmonary tuberculosis is a frequent sequel.

6.—**Trauma and Shock.** Accidents of every kind, railroad, automobile, after operations, prolonged anaesthesia, etc.

7.—**Conjugal Tuberculosis** due to pregnancy, protracted lactation, frequent miscarriages, etc.

8.—**Contact Cases.** This is most infrequent, although cases do occur which may be traceable to intimate or close contact with a tuberculous, followed by infection and disease, however, these are extremely seldom.

Hereditary Factors. Other predisposing factors, some of which will be described more in detail elsewhere, may be mentioned here: For instance, (1) chest deformities; (2) a small upper aperature of the chest; (3) the predisposition in twins; (4) the youngest child in a large family; (5) premature children; (6) children born of parents with too great a disparity in their ages; (7) children of the diabetic; (8) all in whom there is a defective development of the chest or an improper functioning of the various tissues and organs of the body. A small heart, imperfectly developed blood vessels, such as a congenital disease of the aorta or pulmonary (stenosis) artery; (9) a disposition to demineralization in some individuals may be a predisposing factor and this deficiency of lime salt in the organism often constitutes a tuberculous disposition. This we find present more or less in all manifest tuberculosis as well as in pregnancy and in the diabetics giving a high tuberculous hyperreceptiveness. Occupation and race may also play a leading role.

Other Noticeable Tendencies or Disposition. It is worthy of note, at this point, that in young life the glands, the bones, the joints are frequently tuberculously diseased, and this is usually to the exclusion of other organs, whilst in adult life the lungs are often the only organs which are tuberculous. This most likely depends upon the fact that in either the glands or in the lungs at these different ages there exists a heightened tendency to tuberculous disease in these organs. This is a natural tendency, a "physiological disposition" and then again in those instances which are occasionally encountered in which by either lymph or blood stasis or by means of insufficient aeration of pulmonary tissue, tuberculous disease may be brought about. This is often referred to as a "mechanical disposition."

Tuberculosis when introduced into primitive races, like all other germ infections in these peoples is generally followed by a most disastrous consequence, the disease usually running a very malignant course. In

these an inherited disposition is not necessary, the bacilli gaining access upon strictly virgin soil and usually running a very rapid course.

As an inherited disposition plays a great part for the tuberculously infected and the acquired disposition for the tuberculously diseased, our aim must be to decrease this latter disposition, and if diseased, attempt to bring about an arrest of the process, increasing the body's resistance along the line of hygienic principles.

In conclusion let us reconsider for a moment the meaning of these terms, predisposition, diathesis, etc. If the terms as expressed all point to a tissue weakness, a want of body resistance to the implantation of the tubercle bacillus, a favoring of the bacillary growth, would not the words, "a favorable soil or a tendency" be most expressive and synonymous? Or perhaps may not the want of a proper protective epithelial lining of the bronchial tree and which is demonstrated frequently to exist in the early days of infant life in the intestinal tract, favor the entrance of the tubercle bacillus into the organism and be erroneously referred to as the disposition or the diathesis?

CHAPTER 5

INFECTION AND CONTAGION

Infectious and contagious disease (10) or more appropriately, communicable disease.

Note the definition as given in the various medical dictionaries (11):

Infection: The communication of disease from one person to another by contact, either mediate or immediate. (12)

Contagion: The communication of disease by mediate or immediate contact. Communicated by direct or indirect contact.

The words "Infection" and "Contagion," however, are not synonymous; they seem to have a different meaning. May we perhaps assume that the word infection as we use it refers more to the implantation and subsequent growth of the germ and that contagion really relates to the disease as it is? A chronic disease like tuberculosis may be said to be transmitted more or less, by indirect contact, and hence be called infectious, while an acute disease may be transmitted more often by direct contact and so may be called contagious or better both infectious and contagious.

Tuberculosis as we observe it clinically is a communicable disease; it is spread from man to man, or more correctly, from a tuberculously diseased adult to infants and small children with whom the so diseased individual may come in contact.

Tuberculosis is rarely, directly communicated from one adult to another.[1] For this to take place would require the most intimate contact; hence it may be stated that tuberculosis is highly

(1) It must be conceded, however, that under certain conditions adult infection and disease may and does take place. First, the individual who has arrived at adult life and was never infected may become both tuberculously infected and diseased while intimately associating with an active tuberculous person. Here the spreading of the disease to an adult is very similar to that which is observed when primitive people come in contact with the virus. Second, the adult who at some previous time was tuberculously infected perhaps diseased as well, but who for some length of time has lived in an atmosphere entirely free from all tuberculous surroundings, and has for the time being completely lost his immunity may on returning to his former tuberculous haunts, rapidly reacquire the disease and succumb to its ravages. Third, the tuberculous person only mildly infected may on coming in contact with an active tuberculous adult become massively or very virulently reinfected, and having but little resistance develop a rapidly progressing pulmonary tuberculosis.

infectious as well as contagious to infants and small children but not so to adults.

Tuberculosis, like syphilis and leprosy, can not be acquired simply by being in the presence of a tuberculously diseased individual, as in the daily intercourse about the house, meeting on the street, in the workshops, in a street car, in an automobile or at social, religious or political gatherings. As previously stated, to bring about both infection and disease would require the most intimate association.

Tuberculosis, unlike the exanthemata, small pox, scarlet fever, measles, etc., can not be acquired by paying a visit at the home of or by eating, occuping or sleeping in the same room or house with a person suffering from the disease.

Tuberculosis is purely a communicable disease in the young, but it is not contagious to adults in a strict sense. The effect of shaking hands with an actively tuberculous person has frequently been made the subject of special study and usually with a negligible result. Further observation has also proven that on the hands of children living in tuberculous homes and surroundings in only about 10% was it possible to find the tubercle bacillus notwithstanding the fact that in more than 50% of such cases it was possible to demonstrate the presence of the Koch's bacillus on the floors. Tubercle bacilli which are expelled by the diseased person in the act of coughing, sneezing, hawking or spitting are very frequently found in the living rooms about the walls, curtains, bedding, clothing, draperies, etc., and most particularly about the floors, and small children in the immediate vicinity may be directly infected in this way. However, these factors all play in adult infection but a minor role and usually do not lead to tuberculous disease.

Even the probability of both infection and contagion to adults, and perhaps children as well, by moving into houses or rooms previously occupied by a consumptive must always be small, is considered doubtful by some and entirely denied by others. From the known fact that such rooms and dwellings are almost always renovated and cleansed after the consumptive has vacated and before the new tenant takes possession makes this mode of contagion very doubtful.

A tuberculous infection, as has been stated (114), is usually in infancy or early child life, and the frequency of this infection in the first year of life is only about 2%, reaching more than 90%

at about the 14th year or at puberty. This infection is generally followed by manifest disease in nearly all infants during the first year, in 50% of children up to the third, 20% in the sixth or seventh and only about 2% at the age of 14; even after the age of 6 the infection is very seldom followed by active disease, and this because there is in the child at all times a strong tendency to healing. In early infancy the infection generally takes place more readily because the young organism can not protect itself against the infection, and many children who show a tendency to pulmonary disease at the time of their birth, have from birth a constitutional anomaly, a disposition which greatly favors the tuberculous infection. A primary or first infection due to the defence agencies induced by this first infection will give protection to the infected organism from subsequent invasion provided that the later invasion is not too massive nor too frequent.

As nearly all primary infection takes place in infancy or early childhood and usually follows an implantation of the disease germs from without, a later or superinfection generally occurs in youth, or in young men and women. This superinfection is most frequently an autogenous reinfection, that is from within. This generally leads to active disease which is usually brought about by poor nutrition and a vicious life, or by overstudy and overwork, by grief and worry, by disease and trauma, etc. Most adults have been tuberculously infected in childhood, and in consequence thereof have acquired various degrees of immunity and a hypersensitiveness to tuberculin. Such individuals are, as a result, extremely seldom reinfected even in closest association with a tuberculous person, and never during the contact with a disease carrier. It has never been sufficiently recognized by the medical profession that in the adult the contagiousness of tuberculous disease in a strict sense is not a reality, that it is only a theoretical expression or construction and that it is at variance with and contrary to all observations. (Note Saugmann's Critical Study.) No proof is forthcoming that attendants, nurses or physicians to a tuberculosis sanatorium, or those in attendance upon the unfortunates and sick have contracted the disease, and such as have become tuberculously sick were undoubtedly tuberculously infected, perhaps slightly tuberculously diseased, before entering upon the services at such institutions. In our calling as physicians we come in constant, daily and hourly contact with both the tuberculosis carriers and the virus and do we

find the tuberculosis mortality greater amongst doctors than amongst those in other callings?

In the married, tuberculosis of both husband and wife at the same time is very infrequent, notwithstanding the fact of closest contact, the infection possibility here being undoubtedly always present and not to be denied; however, from observations we must conclude that the probability is not very great. We all know of cases of tuberculosis in families where either the husband or the wife is suffering from pulmonary tuberculosis, but with how many cases are we familiar where both the husband and the wife are suffering from active disease at the same time?

The estimated percentage of this family disorder is between 6 and 8, and if we eliminate from this the percentage of men and women who have been infected or perhaps slightly sickened before marriage, it leaves the percentage of those who are infected after marriage, say the husband from the wife, or the wife from the husband, as only about 2%. It is of interest to note that at this time in life the degree of immunity which may be acquired is much greater, is much faster than in child life; as evidence, we may be reminded that if either partner is removed from the environment, is sent either to a sanatorium to take the cure, or is called away by death, the remaining partner heals readily and gets quickly well. In this connection it may also be noted that if either husband or wife develop active pulmonary tuberculous disease soon after marriage, that all the children born will be tuberculously infected and most all will in after life become tuberculously diseased; if, however, in the adult the disease manifests itself later in life when some of the children have been born, then all the children who have passed the sixth year will be spared.

From what has been said, we can conclude that tuberculosis is highly infectious and perhaps contagious to children, but in a sense, it is not contagious to adults; hence the frequent visiting of friends and relatives at the home of the tuberculously diseased person or at the tuberculosis sanatorium should be encouraged. It is wholly devoid of all danger from contagion to adults, and most of all such visits give much encouragement to the unfortunates, are conducive of much improvement in their health, ease the tuberculously diseased person's mind, and they are most grateful to observe that because of their misfortune they are not treated like an outcast and not shunned by fellow man.

As a résumé, we can truthfully conclude that to the healthy adult there is usually no danger in associating with a tuberculous individual,† but it can not be too strongly emphasized that the greatest danger lies in the surroundings of the tuberculous, their homes or otherwise, to infants and small children; hence, the watchword must at all times be, "Keep the small children away."

Dr. Lawrason Brown, Lake Saranac, New York, only recently conducted a series of most interesting experiments at the Trudeau Tuberculosis Sanatorium (56) which may be of note at this point. He first showed what effect room dust, collected from the tuberculous wards, would have on test animals, next the effect of washings from the table utensils, like knives, forks, spoons, cups, etc., from tuberculous patients, washings from tooth brushes, from door knobs, etc., even what effect the shaking of hands of an actively tuberculous person with a non-tuberculous would have. By these experiments he could prove what so often had been proven before, that infection by these means is very infrequent. His most ingenious and unique experiments were conducted to see what effect kissing had on the spreading of tuberculous disease and he concluded from most accurate observations that kissing by an active tuberculous individual with positive sputum in the early morning will undoubtedly spread the disease but that kissing done as the day goes on becomes gradually less and less a source of danger and that towards evening kissing by the tuberculous is entirely free from infectiousness. Positively pulmonary tuberculous individuals, with positive sputum findings, were asked to kiss sterile petre dishes at various times during the day, the washings of these were at once injected into guinea pigs and from this experimental work he observed that the washings from the early morning hours were all positive, the findings becoming less positive towards the afternoon and entirely negative from the washings of the sterile petre dishes kissed in the evening. See Transactions National Tuberculosis Association, Atlantic City, 1919.

Any one who is doing much tuberculosis work and who has treated many cases of pulmonary tuberculosis and whose experience extends over many decades will not have failed to observe the infrequency of tuberculous disease of more than one adult in a given home at the same time. I have observed many cases of tuberculosis in homes where the patient and faithful wife has nursed and taken care of a sick husband for years, and yet in spite of the wife's close confinement to the home, her

†The implantation of the tubercle bacillus into or under the cuticle is not sufficient to bring about infection. The so deposited micro-organisms may remain quiet or dormant for months, in no way make their presence known, and, during all this time it can not be said that the individual is infected. The time from implantation to actual infection may vary from six to eight days to three months. Even if the deposited bacilli should slowly multiply and spread into contiguous tissue we may still speak of non-infection if their presence in the organism has caused no reaction, no tissue disturbance. If, however, their presence becomes known to the body cells, the body raises a defense, protesting against the growth and spreading of the invaders, then we speak of the body's defense powers and that the body is now infected. The moment the resistance begins infection also begins. A tuberculin application is always negative so long as the body's defense powers have not been aroused, the body is then still not infected but with arousing the infection go hand in hand.

companionship and her most intimate association, she did not contract the disease nor at any time show evidence of tuberculosis. Exceptionally I have observed a good and kind husband wait on his sick wife after coming home from work, having his rest broken and disturbed the greater part of the night all without a murmur, perhaps occupying for months before the wife's death the same bedroom or sleeping quarters, and this same man, now years since the death of his wife from pulmonary tuberculosis enjoying the very best of health. Again, a kind and affectionate sister takes into her household a worthless but unfortunate brother suffering from pulmonary tuberculosis, exposes her own children to the infection but never herself shows any symptom of disease. I can well recall the instance of two sisters living for many years in closest companionship, one of the sisters developing a pulmonary tuberculosis, and though from the onset of the disorder until death more than thirteen years have elapsed, these two sisters during all this time were inseparable, the early years and the greater portion of time being spent in travel and at sanatoria seeking the cure. The last year of the sick sister's life was spent entirely in bed, and during the greater portion of all this time the healthy sister shared the same bedroom, to be at her side, to serve her every want, to make her last hours comfortable, and after all these years of sacrifice, deprivation and devotion, upon the death of the sister, the remaining one was in perfect health. The case of two adults in the same household, the one becoming sick upon the attendance of another individual sick with pulmonary tuberculosis, must be exceedingly infrequent. In all my years of experience I have never observed such a case, hence I ask how many cases do we see where both husband and wife are suffering from active pulmonary tuberculous disease at one and the same time?

CHAPTER 6

THE PRIMARY FOCUS OR FOCI OF INFECTION MOST FREQUENTLY IN THE LUNGS AND THE SUBSEQUENT AFFECT ON THE PULMONARY TISSUE ([1])

As early as 1876 Parrot (15) reported from numerous autopsy observations on infants and small children that in early infancy, in every case of pulmonary disease, tuberculous as well as non-tuberculous, a simultaneous involvement of the regional lymp nodes can be demonstrated, and, vice versa, that no change in the lymph nodes about the lungs can be shown without an analagous involvement of the lungs; that a tracheobronchial lymph gland involvement is evidence of pulmonary disorder and that a primary focus is invariably to be found somewhere in the pulmonary tissue. This became known as Parrot's Law, "LaLoi des adinopathie similaires," and it applied with equal force in all cases of pulmonary tuberculosis.

Parrot, on 145 autopsy observations on tuberculosis in children, was always able to demonstrate in the presence of a tracheo-bronchial lymph gland involvement, a primary focus at some point in the lung, "Le foyer pulmonaire primitif." Hervouet, a student of Parrot, on further observations states that the changes in the lungs are not always accompanied by a similar change in the regional glands, but that if changes in the glands are demonstrable, always and invariably similar changes will be found in the lungs. However, the most important and definite observations were those of Küss in 1898. He confirmed the existence of Parrot's Law and described in minute detail the portal of entry of the tubercle bacillus into the infected organism. According to Küss the primary invasion is characterized by a small nodule situated usually subpleurally, in which condition it may remain for years, gradually undergoing retrogressive changes, which are so frequently observed in tuberculosis, or it may ulcerate, the nodule soften and its contents

[1]Under this chapter we will consider only the infection as it is brought about in the natural way, leaving artificial inoculation to experimental medicine.

may be emptied into a bronchus. In this condition the regional lymph nodes are mostly always secondarily involved, showing a definite connection existing between the tuberculosis nodule in the lungs and the nearest glands. In most instances but a single primary process was demonstrable; two or more foci were only occasionally observed and the lymph tract between these primary pulmonary foci and the regional glands remained usually in a healthy condition. According to Küss, this primary pulmonary focus frequents both the right and left lung but favors in most pronounced form the lower lobes, is occasionally found in the upper, but never at the apex. From this he concluded that the current of air is capable of carrying the bacilli along the

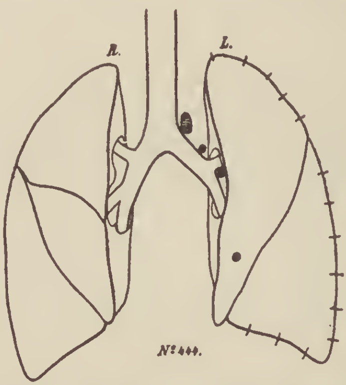

Fig. 1. The lung (schematic) after Ghon. Autopsy No. 444. Cause of death tuberculosis meningitis, boy 7 years. Note the calcified nodule, in the lower portion of the left upper lobe, the lingula pulomalis, surrounded by fibroid induration, this the initial infection, the single primary focus. The regional lymph nodes the upper left broncho-pulmonary and tracheo-bronchial have undergone change, both calcification and caseation. The cross lines along the border of the left lung are indicative of pleurisy.

bronchial tubes, but that they are usually deposited there where the current of air is vigorous and most direct.

All this labor was later still more emphasized by E. Albrecht who in 1907 showed that in right sided pulmonary involvement, the right tracheo-bronchial glands, and in left sided disorder, the left regional lymph nodes become secondarily involved. He states further that in every case of tuberculosis of the bronchial glands, the corresponding nodule in either right or left lung should be looked for. H. Albrecht, 1909, studied the primary foci in 1,060 observations on children at autopsy, arriving at similar results.

The most interesting, definite and conclusive labor done in recent years is that of Ghon, who in 1912 made known his ob-

servations on 184 carefully conducted autopsies in tuberculous children. He divided this number of cases into two groups, the larger or 170, in which he was able to demonstrate uncontrovertably a primary tuberculous focus in the lung, and the smaller group of 14 cases in which he was not able to demonstrate positively such a focus, although conditions existed in the pulmonary tissue which may presuppose such a focus. He further proved that in 142 cases out of the total of 170, only a single primary focus was present; in 15 cases two foci could be shown, in 5 cases three, in 2 cases four, in 1 case five and in 5 cases an indefinite number of minute foci. According to his observations, primary infection of the right upper lobe predominates; next in frequency is the left upper, followed by the right and left lower, and the middle lobe was found eleven times to be the seat of primary infection. It is worthy of note, that in nearly all of his observations, if the right or left lung was the seat of infection, the primary seat of the infection was to be found not in any portion of the lung tissue, but chiefly near a bronchus, but not in a bronchus, and only very exceptionally toward an apex either right or left, that is, the seat of primary infection was usually found near the middle portion of the lung. Ghon found further that the right lung was more frequently the seat of primary infection as compared with the left, and that the right upper lobe was out of choice the lobe involved. This, however, is contradictory to the findings of Küss, according to whom a primary focus favors the right or left lung with equal frequency, but most particularly selects the lower lobes.

In Ghon's report of 184 cases, we find that the youngest tuberculous child coming to autopsy was 2 months and the oldest 14 years of age, and in addition, we find that most frequently the lower lobe is the seat of primary infection in the younger and the upper lobe in the older children.

Perhaps the observations of Küss were upon a greater number of smaller children, hence the infection was found more frequently in the lower lobes. The question now arises as to why a primary focus should be localized, particularly in infants, more frequently in a lower lobe as compared with an upper. As Küss has already pointed out, and reference has already been made to this, the air current through the bronchial tubes is capable of carrying the tubercle bacilli along any part of the bronchial tree but they are deposited most frequently along those sur-

faces where the current is most pronounced, vigorous and direct. If this is true, and this has not been disproved, then the implantation of the tubercle bacillus either as a primary involvement or as a subsequent disease is simply a mechanical process, similar in both man and animals. This also explains the overwhelming frequency of tuberculous disease in the upper lobe in the adult and the right lobe more so than the left (65 to 75% right apex disease and 35 to 25% the left). This will also readily explain the relative pulmonary tuberculous disease in the bovine and other quadrupeds. In the human adult suffering from ac-

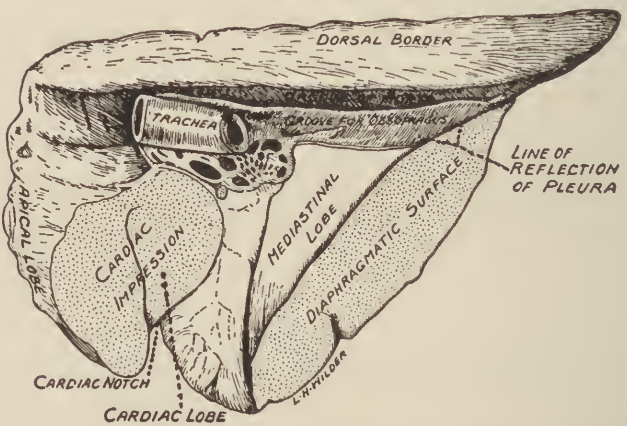

Fig. 2. Lateral view of the bovine lung. The shaded portion, the tip indicates the most frequent point of election in tuberculous disease. In the bovine the apical lobe is also frequently involved, the cardiac the most pendent portion very rarely, this even in the last stages of the disease. From Sissons, J. A. M. A., p. 1513—1918.

tive pulmonary tuberculous disease, we usually find the apices infrequently are the bases primarily actively diseased. In small children, however, we quite often find active tuberculosis of a lower lobe as a beginning disorder. This, as in the primary infection, also is brought about by mechanical means.

The tuberculously infected individual is generally reinfected from within and not from without, and this leads to active disease. This is a secondary aspiration process. The bronchopulmonary glands having received the lymph flow from the primarily infected lung become secondarily infected and ultimately undergo either a progressive or a retrogressive change, becoming either soft and caseate or calcify or undergo fibroid changes. During this time the primary focus in the lungs may heal, leaving an indelible scar. If these bronchial nodes which are sec-

ondarily infected should undergo such changes, either calcifying or undergoing fibrosis, they may then remain quiescent for years without any other or perhaps slight retrogressive changes. Should these glands, however, as so often occurs, become caseous and soften with accompanying irritation and inflammation of the surrounding tissue, then the contents of such glands may break through the fibrous capsule, find access into a bronchus through the point of least resistance and entering the lumen again be carried by the inhaled air in the direction of the greater air current. In the adult when in the upright position, this is

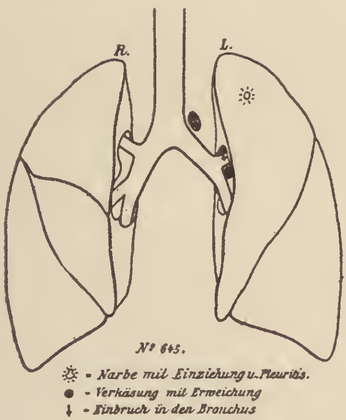

Fig. 3. The lung (schematic) after Ghon. Autopsy No. 645. Cause of death Miliary tuberculosis, male, age 4 years. Note the scar in the left upper, towards the middle of the lobe surrounded by a secondary retraction and pleuritis. The regional lymph nodes, the left broncho-pulmonary and tracheo-bronchial have undergone caseation and softening. Note carefully the arrow showing the breaking through of a softened gland into the bronchus near the hilum. In such an event bacilli in the sputum may or may not be evidence of pulmonary tuberculosis.

usually in the direction of one of the apices of one of the upper lobes, depending upon whether or not the primary infection, followed by the involvement of the regional lymph node, was in the right or the left lung. But Ghon has shown that out of a total of 142 cases of pulmonary involvement, a single primary focus was demonstrable in the right lung 87 times or 61.27% as compared with the left 55 times or 38.73%. This goes to show that the right lung is most frequently the seat of primary infection, and that the regional lymph glands become secondarily infected, that the nearest glands to the right lung are the lymph nodes to the right of the bronchus and that subsequently it is the glands to the right that undergo changes which are ultimately followed by right sided pulmonary disease. If the left

lung is primarily infected, the lymph nodes adjacent to the left bronchus become secondarily infected, and this is later followed by active tuberculous disease of the left lung.

In order to comprehend clearly how primary infection and later active pulmonary tuberculous disease takes place through the respired air, it will become necessary that we first consider the anatomical structure of the bronchial tree from its origin at the trachea down to its minutest ramifications in the pulmonary tissue, namely, (1) main trunk, (2) primary bronchi, (3) secondary bronchi, (4) tertiary bronchi, (5) the bronchioles, bronchi respiratori, (6) the terminal bronchi, the alveolar ducts, ductuli alveolares, and (7) the atria (vestibuli), the air sacs and alveoli (sacculi alveolares et alveoli pulmonum), these latter being the last division within the pulmonary parenchyma.

Beginning at the larynx, which may be likened to the underground portion, the root of a tree, the trachea or main trunk extends downward, inclines a little towards the right and at a point on the anterior chest wall opposite Ludwig's angle, or perhaps a trifle lower, on a line with the second interspace, and posterior on a level with the fourth dorsal spine, divides into two lesser trunks, the primary bronchi, one to the right, the other to the left, continuing to the hila of the lungs which they enter and almost immediately begin to again divide and subdivide into smaller and smaller trunks, known as secondary and tertiary, and extending like the branches of a tree in all directions, terminating towards the pulmonary periphery in most minute tubules known as the respiratory bronchi, at the distal ends of which and now in the pulmonary parenchyma are found the alveolar ducts and the air vesicles (59).

The trachea, the bronchi (23), the various branches, both the secondary and the tertiary are not continuous straight tubes,[2] which become smaller and smaller as they approach the pulmonary periphery, but on the contrary immediately below the larynx the trachea is somewhat contracted, becoming wider as it extends downward and again contracting as it approaches the bifurcation, at which point it again widens, continuing in this

[2]Prof. W. S. Miller, of the University of Wisconsin, has conclusively demonstrated that the continuation of the bronchioles is not abruptly into the alveoli, but that their terminals are most complex bodies usually dividing into many compartments or air sacs, breaking up and terminating in the pulmonary parenchyma, that between these air sacs and the respiratory bronchi or bronchioles, we find the vestibules or atria, these in turn opening into minute ducts, the ductuli alveolaris, the terminal bronchi, the continuation of the bronchioles.

manner throughout the primary bronchi and to some extent the secondary and tertiary branches. By this anatomical structure the respired air entering is given a distinctly rotary motion. See Figure 4

If we outline on the chest wall the course of the bronchial tree and its various branches we will observe that the latter

Fig. 4. Representation of a Primary Lobule of the Lung (Schematic). An Anatomical Unit—After Prof. W. Snow Miller, University of Wisconsin. Bulletin, Chicago Tuberculosis Institute 1916.

EXPLANATORY NOTE

b.r.—A bronchial (bronchiolus respiratorius).
d.al.—An alveolar duct (ductulus alveolaris).
a.—An atrium (vestibulum).
a.p.—An alveolus (alveolus pulmonis).
s.al.—An air sac (sacculus alveolaris).
p.—The pleura. The lobule is represented as being situated immediately under the pleura.
x.—Ring of muscle fibre. End of all muscle tissue, lymph vessels and nucleated epithelial cells. (See Alfred Stengel, Nothinagel's Practice.)
y.—Represents the beginning of a second primary lobule, which is, however, not carried out in detail.

become extremely small, terminating posteriorly on either side of the spine on a level with the eighth rib, anteriorly on the left side at the fifth rib internal to the mammary line, right side, fifth rib near the parasternal line, and all the tissue beyond and towards the lung border is occupied by the pulmonary parenchyma, the minute divisions of the distal ends of the termi-

nal bronchi. If next we follow along the bronchial tree, from the main trunk at the larynx down the primary bronchi, the secondary and tertiary branches to the terminal bronchi, the distribution of the muscular tissue, smooth muscle fibers, we will observe that all of this tissue becomes gradually less and less in amount as we proceed downward and outward approaching the pulmonary parenchyma, and that all muscle fibres have entirely disappeared, with perhaps the single exception at the terminal bronchi, where a ring of unstriped muscle fiber, sphincter-like, guards the entrance to the atria at the distal ends of the ductuli alveolares. No muscle fiber is found beyond this point or in the pulmonary parenchyma, all muscle fibers stopping at the distal ends of the terminal bronchi. Now if we follow the bronchial lymphatics, which form a close net-work or mesh-like structure in the walls of the bronchial tubes, we will observe that they diminish in size as we proceed from the hilum pulmonum, becoming smaller and smaller, and terminating with the terminal bronchi, and that no lymph vessels are found beyond this point, that is, beyond the ring of unstriped muscle fibers and that there are no lymph vessels in the walls of either the air sacs, atria or the alveoli, all of which are situated in the pulmonary tissue proper. Let us now consider for a moment the third important factor in this anatomical picture, the epithelial lining of the bronchial tree from the main trunk to its ultimate termination in the alveoli or air sacs. From and including the larynx, the trachea, the primary bronchi, as well as the secondary and the tertiary branches and as far as the respiratory bronchi, we find epithelial cells of the ciliated cylindrical variety lining these tubes, but on the walls of the terminal branches of the bronchioles, the alveolar ducts, we find a simple epithelium of the cuboidal variety, and these cells of cuboidal epithelium extend towards the distal ends, gradually becoming flatter and ultimately joining at the atria the epithelium of the flat pavement variety, polygonal cells, which line the atria, the air sacs and the alveoli.

As in a tree not all the leaves are found at the outer border but many arise from smaller branches nearer the main body, so in the bonchial tree all the air sacs do not arise at the periphery, but many may arise in clusters about the smaller and the terminal bronchi. These are, however, found to be smaller and less numerous, though the general structural arrangement throughout is everywhere the same, and they are lined with

the same flat pavement epithelium as the larger and more numerous air cells.

If next we study closely the structure of the cells lining the bronchial tree, beginning at the larynx and continuing up to and including those which line the terminal bronchi, we find that they are all of the nucleated variety, both the ciliated cylindrical and the cuboidal, but that those cells which line the alveoli, the atria and the air sacs differ in being of the non-nucleated kind.

Loeb (208) in 1899 advocated the idea that the nucleus of a cell is its center of oxidation, is the life of the cell, and that this will explain why cells without, or being deprived of, a nucleus are very short-lived and very easily destroyed, show no resistance and are wholly unable to regenerate missing parts. Matthews (209) in 1915 stated that the nucleus in plant cells is directly concerned in the oxidation process, and Spitzer (210) as early as 1897 reported that the nucleoprotein extract from certain animal tissue has the same oxidizing power as the tissue itself. From these observations we can conclude that the nucleus of the cell in both plant and animal tissue possesses highly oxidizing properties, a power which is not possessed by the non-nucleated cells, and moreover, that the non-nucleated cells are short-lived, are easily destroyed, show no resistance and are wholly unable to regenerate missing parts. Now, the cells lining the air sacs, the terminal and distal ends of the bronchi within the pulmonary parenchyma are all of the non-nucleated variety, hence possess very little, if any, resisting power and consequently must be very readily destroyed by any foreign body entering the cells. From pathologic study we know that pulmonary tuberculosis, both the primary infection and later the disease, has its origin, not in any part of the lung, not in the bronchial tubes, but in the pulmonary parenchyma surrounding the smallest bronchi, namely, in that portion of the lung in which the air sacs are situated and which are lined with non-nucleated cells.

Parenthetically it may be stated that the assertion that free openings or stomata lead from the alveoli, the atria and air sacs or air cells into the lymph vessel of the lungs has not been confirmed, and is very improbable, as lymph vessels are not demonstrable in the walls of the alveoli nor in the atria.

Having considered somewhat in detail the anatomical structure of the bronchial tree, let us now consider how tuberculous infection has its beginning in childhood, and subsequently be-

comes active in adult life. It is necessary that we consider at the onset all the important factors, namely, the air currents, the bronchial tree, the muscle fibers along this tree, the network of lymph vessels in the walls of the different branches of this tree, the epithelial lining of these tubes and particularly those which line the walls of the terminal ends, the air sacs, the non-nucleated cells, and above all the tubercle bacillus entering the bronchial tubes with the respired air.

The air on entering the main trunk is thrown into a rotary motion, this whirling motion is continued throughout the whole bronchial tract and particles of foreign matter, like minute specks of dirt, coal dust or bacteria, are in this manner readily carried along with the current. However, the greater portion of these foreign particles is again expelled by the motions of the ciliated epithelium lining the bronchial tubes and by the contractions of the muscle fibers accompanying them throughout, still particles of dust, and tubercle bacilli as well, do find lodgment on the bronchial mucosa, and some may gain access by penetrating the irritated mucous layers, and if so, they are in all probability taken up by the close network of lymphatics lining the entire inner walls of the bronchial tubes, and as all of these lymph vessels drain towards the lymph nodes in the hilum, the regional glands of the tubes, particles of dust, coal and tubercle bacilli among others are deposited in these glands. This corresponds to the findings of Küss who showed that the lymph tract between the primary foci and the regional glands usually remained in a healthy condition.

That tubercle bacilli are not deposited within the walls of the bronchial tubes per se is evidenced by the fact that a primary tuberculous infection and a secondary reinfection as well are never found in these tubes. We must, then, assume that some, or at least a few tubercle bacilli, after passing the ring of muscle ([3]) fibers, are carried onward and outward towards the pulmonary periphery into the distal ends of the terminal bronchi, where they find prepared a portal of entry, a nidus for their deposition and further growth.

All lymph flow in the pulmonary tissue is towards the peri-

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[3]That these rings of muscle fibres placed at the terminal ends of the bronchial tubes really guard the entrance to the air sacs can fairly well be demonstrated from observations which have so frequently been made, namely, that when otherwise healthy coal miners are accidentally killed while at work, autopsy will show the bronchial tubes filled with coal dust, but the air vesicles will be found comparatively free.

bronchial glands.[4] These become secondarily infected whilst the primary focus in the lung tissue often heals entirely, leaving a typical scar, often only pinhead in size—the indisputable evidence that this was the primary or the older infection. It is well to remember that the first point of tuberculous infection is not an involvement of the pulmonary lymph nodes, such gland structure, if infected, being always secondary to a primary seat in the adjacent pulmonary parenchyma. Post mortem evidence has sustained these findings, namely, that the pulmonary tissue is always first infected, and that an involvement of the pulmonary or peribronchial glands is a subsequent infection.

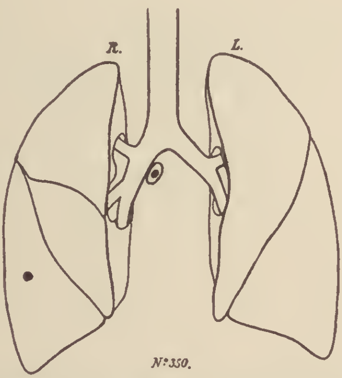

Fig. 5. The lung (schematic) after Ghon. Autopsy No. 350. Cause of death scarlet fever, girl, 7½ years. Note the calcified nodule, the initial lesion evidence of an old healed process in the middle portion of the right lower lobe with secondary calcification of a single regional lymph node situated in the lower right tracheo-bronchial area.

In this connection it is interesting to note that both Küss and Ghon have observed that in the greater number of instances only a single primary focus is found in the lung tissue, that occasionally two foci are found and that three or more are exceptional, all of which goes to prove that in the human, a natural infection is very similar to the artificial inoculation produced in the integument of animals where after a primary infection a subsequent reinfection is usually negative.

After the primary foci are healed, the peribronchial, the glands nearest to the point of first infection, are found to be secondarily involved. (See Fig. 5.) If this first infection has been mild or

[4] It has been repeatedly demonstrated that when live virulent tubercle bacilli are placed into the alimentary canal, for instance into the large bowel, that infection of the peribronchial or hilus glands may take place without previously involving the healthy lung structure but this is an artificial infection in an experimental animal and not the natural infection which we are now considering.

at any rate not massive, then these glands gradually undergo retrogressive changes and a period of quiescence manifests itself, in many instances for years ([5]), the lymph nodes becoming more and more encapsulated or undergoing calcification, no further disorder being noticeable. In many instances, however, after such a period of either short or long quiescence, the bacilli again become active, the glands undergoing swelling and softening and ultimately expelling their contents in the direction of least resistance. In some instances this point of least resistance is in the tissues about the bronchial tubes or in the mediastinum, where it is quickly taken up by the lymphatics, producing a generalized or a miliary tuberculosis, or it may break into the esophagus and the contents find their way into the alimentary canal, where the fate of the bacillus depends on the quantity of bacteria which enter the abdomen and also upon their virulence. In other instances, the focus of least resistance is through a bronchial tube (see figure 3) from which the contents of the gland may simply be expectorated, healing of the lacerated gland and the open bronchus taking place, and here also nothing further being noticeable. But as frequently happens, the contents of such a gland either in part or a small quantity at a time, may find entrance into a bronchus, be aspirated along a bronchial tubes with the currents of air and ultimately find again a nidus now in the distal ends of the terminal bronchi, in the air sacs, the alveoli and infecting or better now reinfecting from within, the pulmonary parenchyma, this, however, is unlike the first, and is usually not followed by scar tissue formation but by active tuberculous disease, and tissue destruction. This now is the condition described as pulmonary tuberculosis, a secondary aspiration process, a disease situated about the terminal bronchi and the air sacs or alveoli, the distal ends of these tubes, ultimately producing a peribronchial infiltration.

Why the tubercle bacillus in primary infection, in child life, should show a special predilection for the pulmonary parenchyma of the lower lobes (88), and in the adult, the secondary or re-

[5]That a long period of quiescence often follows the implantation of the tubercle bacillus, that sometimes years elapse before manifestation of the disease, is now generally accepted. We find, however, even in the medical profession, that this is often taken somewhat skeptically, hence the enumeration of a few physiological instances. Histology teaches us that in the upper lip of the male child the roots from which the moustache grows are already present at birth, and yet it takes the stimulus of puberty to make them grow. Our dentist friends inform us that the roots from which the wisdom teeth grow are to be found in the jaw of the child at birth and often as much as 28 years have elapsed before they are erupted. Better still, from Gynecology we learn that in the female ovary the roots or germs of many thousands of ova are demonstrable at birth, and yet in many instances a Graefean follicle does not ripen until 45 or more years.

infection select the upper lobes can readily be accounted for when we consider the postural positions in these different years or ages of life. Küss, as early as 1898, and this was later corroborated by Ghon, pointed out that the primary infection in the lungs in children is in the direction of the greatest and most direct air current, and undoubtedly the same applies to adult life and to animals as well. In cattle the point of pulmonary tuberculous disease is usually found in that part of the lung which corresponds to the human, in what is generally known as the caudal portion—the tail end, that is, the base. This, in the animal, is also a secondary infection from within, an aspiration

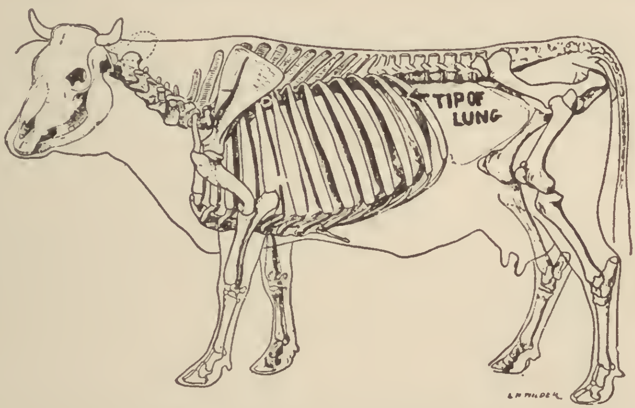

Fig. 6. The Lungs in Situ in the bovine. The arrow points to the point of election in tuberculosis which is in the tip of the lung and which is also the highest point whether the animal is standing or lying. This part of the lung corresponds with the base or caudal portion in the human. From Sissons J. A. M. A. p. 1512—1918.

process from a large, previously infected gland pouring its contents, laden with bacilli, into a bronchus and being carried by the air currents to the finest but most vulnerable tissue of the lungs. We note here also, that the current of air is in the direction of least resistance.

It now becomes apparent why different parts of the lungs in both man and animal seem to exhibit a special predilection for the tubercule bacillus. In early childhood, in infancy, owing to the usual postural position of the child at that age, which is either lying down or on all fours about the floor in the home of the tuberculous adult from whom it receives its primary infection, the most favorable point for the respired air is towards the base, where the air current has more free access and the same applies, undoubtedly, to the bovine, which throughout the

greater part of its whole life, in its natural postural position, places the base of the lung constantly at its highest point. This all favors a more direct air current and it is there that we find both primary infection and later active tuberculous disease. In the human, in adult life, the secondary or reinfection is in most instances towards the apices. It is there that active tuberculous disease first manifests itself, again in the direction of the greatest air currents, which in the upright postural positions during adult life form a more direct, a more easily accessible route, a more ready way to carry the aspirated tubercle bacilli mechanically upwards and outwards, into the most delicate and most vulnerable pulmonary tissue.

Active pulmonary tuberculous disease begins as an alveolitis and bronchiolitis. Aspirated tuberculous material from a softened gland is carried with the strong air current directly up into the pulmonary structure, along the bronchial tubes and alveolar ducts into the alveoli. There it meets a cellular lining, an epithelium which offers no resistance, is easily destroyed, cannot protect itself or show any defense, and the contained bacilli—like all invaders—take possession of this delicate, defenseless tissue, begin the preparation of a new soil for their growth and development and little by little penetrate deeper and deeper into the contiguous tissue, surrounding more and more bronchioles and alveoli, gradually producing the morbid process in the lungs described as peribronchial tuberculosis, the subsequent growth and development, rapidity or retrogression, depending upon the fertility or non-fertility of this newly invaded soil, and upon the environment and age of the tuberculously affected individual.

Recapitulation

1. The bronchial tubes are accompanied by smooth muscle fibres throughout; at their distal ends all these fibres cease but for a ring of unstriped fibres which guards the entrance into the air vesicles.

2. The walls of the bronchial tubes are lined throughout by a fine net-work of lymphatics, but these stop abruptly at the ring of muscle fibres situated at the terminal ends of the tubes.

3. Ciliated cylindrical epithelial cells line the entire bronchial tubes with the exception of the alveolar ducts, the terminal bronchi, which are lined with a cuboidal epithelium. Both the ciliated, cylindrical and the cuboidal epithelial cells are of the nu-

cleated variety. They do not extend beyond the ring of muscle fibres mentioned.

4. The epithelial cells which line the alveolar walls, the air sacs, distal to these rings of muscle fibres, are of the polygonal variety. These cells are non-nucleated, are short-lived, are easily destroyed, have no resistance or defense power and can not protect themselves against foreign bodies, like bacteria, dust particles, tubercle bacilli, etc.

5. It is known that on the living, healthy, nucleated epithelium lining the bronchial tubes, the tubercle bacillus is perfectly harmless, and for that reason pulmonary tuberculosis never has its origin in these tubes. The epithelium of the air vesicles, however, are all lined with polygonal cells which are non-nucleated and which are situated beyond the rings of muscle fibres.

CHAPTER 7

THE COURSE OF TUBERCULOUS DISEASE IN THE DIFFERENT AGES—FROM INFANCY TO OLD AGE

Throughout life, at every age and at any time, that is, from early infancy on, up to old age, the tuberculously infected individual may manifest active tuberculous disease, either in one form or another. This tuberculous disease may show a special predilection for certain tissues or organs of the body to the exclusion of others, producing either a localized manifestation or at any time assume a virulent type, invade many organs or tissues and become generalized as is observed in miliary tuberculosis (114).

Tuberculosis (86), a disease entity, as we observe it, usually presents a continually changing picture beginning in the vast majority of cases with a primary infection in early infancy, gradually invading various organs and tissues of the body and terminating in adult life with the fully developed pulmonary tuberculous disorder.

Primary infection usually takes place after birth and is then spoken of as extra-uterine or post-natal in contra-distinction to that infection which takes place occasionally before birth which is known as intra-uterine or pre-natal.

Intra-uterine Infection. Pre-natal, Congenital or unavoidable. A true congenital tuberculosis is a rarity and is very infrequent. Infants born with a tuberculous infection usually succumb early to a generalized tuberculosis (within the first few weeks of their existence) and they very rarely see the second month of life. It is stimated that in only about 1% of all cases the infection takes place intrauterinely. The mortality will always be great because the infant is not only suffering from intrauterine infection but the child is actually born with an already existing tuberculous disease in one organ or another. It is hardly conceivable how infants born with a primary tuberculous infection can carry that latent infection into adolescence without becoming early tuberculously diseased.

Extra-Uterine Infection. Post-natal, Postgenital, avoidable,

or acquired. Most infection, about 99 percent of all takes place after birth, the time of infection depending upon how early or how late the infant comes in contact with the tuberculosis virus, that is, with an active tuberculous individual. As is stated above nearly all tuberculous infection is conveyed from man to man, generally from a tuberculous adult to infants or small children and the earlier in life of the child this infection has taken place the greater will be the early mortality. It is estimated that the number of infants infected in the very early days of their existence is very small, that only about 2 percent of all children born are tuberculously infected in the first few months after birth, but that the mortality of all so infected is usually very great; that all infected in the first few months succumb to a generalized tuberculosis and do not see the beginning of the second year; that, however, the mortality decreases as the number of days between birth and the first infection increases, that is in the first months of infant life and the question of infection also becomes a question of quantity, whether few or many organisms are present, and whether they are virulent of avirulent. If this first or primary infection is massive, that is if many virulent tubercle bacilli enter the infant organism at a given time, or if the infection with virulent tubercle bacilli is oft repeated for only a short period, then no organ or tissue is spared from the invaders and the child succumbs to a generalized tuberculosis and rarely sees the beginning of the second year. The mortality at this period is always great, estimated to be anywhere from 95 to 100 percent.

It should be remembered that between a massive infection when great numbers of tubercle bacilli gain entrance into the infant body and definite and strong symptoms follow, and an infection consequent upon the entrance of only a few bacilli with none or perhaps the most mild symptoms all grades of intoxication and disease may be noticeable. All infants only mildly infected in the first year of life as well as those who are infected after the first, that is during the second and third, either massively or mildly, constitute the great army which in the subsequent years are the victims of tuberculosis in one form or another. In early infancy, and this applies to both those who are infected before birth or intra-uterine, as well as those in whom a massive infection came about soon after birth or extra-uterine, there is a strong tendency to a generalized disease and speedy death; however, this tendency seems to lessen with the age of the

child and up to adolescence when it has assumed a more or less benign aspect, during all these years displaying a special predilection for certain organs and tissues in each decade or semidecade. In the earlier years, that is from the second and up to the 14th the disease manifests itself chiefly in the organs and tissues supplied by the greater circulation, the pulmonary circulation up to this time has been comparatively free from active disease.

Primary Tuberculous Disease. After pulmonary infection has taken place and the regional lymph nodes have become secondarily infected, a period of more or less quiescence generally supervenes, and later from various causes these glands about the hilum and near the bronchial bifurcation, the epibronchial, peribronchial, tracheo-bronchial, broncho-pulmonary and the tracheal as well as the mediastinal are now most frequently not only the seat of tuberculous infection but of tuberculous disease as well. In the child, this is most noticeable in the years up to and including the fifth year when again a decided change is observable.

Secondary Tuberculous Disease. Metastatic, lymphogenous and hematogenous. Two types may be recognized. (1) A malignant, and (2) A more or less slow or more benign form. The malignant is usually hematogenous, the bacilli gaining access directly into the blood stream from a caseous or degenerating gland producing miliary or basal meningeal tuberculosis. The second form most likely lymphogenous—many bacilli after entering the lymph channels are held back by the nearest glands and only few gain access into the circulation. This, however, is then followed by bone, joint, spine and slow meningeal disease.

At this point we observe a strong tendency to a less frequent involvement of the lymph glands but a distinct metastatic tuberculosis of other tissues and organs of the greater circulation; and tuberculosis of bones and joints, the meninges, the skin, spine, etc., are now most prevalent, up to this time more or less sparing the lungs. Now the frequency of bone and joint disease predominates and in the earlier years and up to the 6th and perhaps the 7th year, tuberculosis of the small bones and joints is most frequent, and disease of the wrist and ankle bones, the carpal and tarsal, the metacarpal, metatarsal and phalangeal are now mostly encountered and as the child advances in years and towards puberty, we find that the larger joints, the knee, the elbow,

the shoulder, the hip, etc., all of which until now have been spared from early involvement are most frequently the seat of active tuberculous disease. It is noteworthy at this point to observe that the mortality from all forms of tuberculous disease has lessened, the disease having now assumed a more or less benign form and that at the age of puberty and a little beyond, up to the age of 14, only about 2 percent perish from tuberculosis. As the frequency of infection rises, the mortality decreases and the ratio between the tuberculosis mortality and those who are tuberculously infected is in inverse proportion. In early infancy, the infection, as stated above, is usually estimated to be between 1 and 2 percent with a mortality of nearly 100, whilst at the age of 14 with a mortality estimated at only 2 percent we find that the number of those who are tuberculously infected reach more than 90 percent.

Tertiary Tuberculous Disease. With the beginning of life, that is in the first decade, tuberculosis assumes a very acute form, more subacute towards the middle of the decade and towards the close and beginning of the second a chronic or benign form and in the middle of this decade after the organs and tissues of the greater circulation have stood the brunt of the disease, have now acquired so to speak, a certain degree of immunity, we again notice a decided change in this constantly changing tuberculous picture, for we will observe that from now on the organs of the lesser circulation, the lungs, which until now have been comparatively spared, are most vulnerable and particularly these pulmonary attacks in this the middle of the second decade are like at the beginning of the primary infection most virulent, acute and fatal, becoming subacute towards the close of the decade and during the greater portion of the third, after which, that is during the fourth and fifth, the disease again assumes a chronic and benign form and as compared with the pulmonary tissue now involved during this time, all other forms of tuberculosis are very infrequent.[1]

In old age we again note a variation in the changing picture. We have found that in infancy the great tendency is to a very acute generalized form of the disease; a little later in child life, it becomes more subacute and towards and during adolescence, a more chronic, generalized form. With advancing age, there

[1]It is true that we occasionally see gland, bone and joint tuberculosis in an adult and we also see at times pulmonary tuberculosis in child life but not as a rule—these are exceptions.

is again a great tendency to the generalized form of the disease, now very chronic and much protracted, localized chiefly in the lungs but not in the apices as we find it in earlier life, but anywhere throughout the lungs, chiefly at the bases. In the aged, a lessening of the body resistance becomes evident and the mode of living, sedentary habits, indoor life, much confinement in the homes or in rooms are all factors which tend to reactivate a long and insidious latent process.

It is most interesting to note the tuberculosis mortality during the different periods of active disease. In early infant life the mortality of those who are infected is always high and all who are heavily infected in the first months succumb to the disease within the first year, the estimated percentage of death being anywhere from 95 to 100. At the age of 3, about 50 percent of all tuberculously infected children succumb to the disease in one form or another, 20 percent from the 5th to about the 7th and only about 2 percent at the age of 14. Up to this period pulmonary tuberculosis has been very infrequent, the other tissues and organs of the body being most frequently the seat of tuberculous disease, but we will observe that they seem to have now acquired a degree of immunity and a less frequent involvement, but a very perceptible increase of pulmonary tuberculosis is now apparent and with this increase of disease, an increase of the tuberculosis mortality is evident, which mortality again lessens with each passing decade.

Every child which is tuberculously infected has not been properly protected, hence it becomes necessary to guard against and avoid infection in early life in the young; in youth we must guard against a latent tuberculosis becoming active. The v. Pirquet is positive in only 5 percent of all children at the end of the first year of life, increasing gradually so that at the end of the 5th year about 20 percent react and more than 90 percent at the age of 14. That obtains in all children infected after birth, post-natal, in the prenatal or congenitally tuberculous we do not know at which time the skin reaction is positive; it is usually negative. In these there is no period of latency and the absence of all allergic possibilities is due to the inability of the body cells to produce defense agencies or antibodies.

Resumé. In early infant life a massive infection from contact with an active tuberculous individual is usually followed by great mortality; in later life however, the infection is generally more

mild, is not so massive, although it may be oft repeated, and is then followed by a slow chronic process. In children a tendency to chronic tuberculosis with connective tissue proliferation about the tuberculous process is extremely rare; here there is a greater tendency to a generalized disorder with cavity formation, which in very early life is quite frequent—vomicae having been found in the lungs of infants at autopsy. In children who have passed the eighth year of life the disease assumes a more chronic form like pulmonary tuberculosis in adult life, showing a great tendency in healing. Here, we usually recognize two forms of the disease —(1) a rapidly fatal form, a miliary tuberculosis, with symptoms of meningitis, pleuritis or peritonitis usually with a sub-acute pulmonary tuberculosis persuing a rapid course; (2) a more or less curable form. This may manifest itself in (a) a localized form with tuberculosis of the serous cavities and of the vicera; (b) with slight lesions in the lungs, in joints or in the pericardium; (c) forms without definite localization, such as may simulate typhoid, usually accompanied by peribronchial gland tuberculosis.

CHAPTER 8

HISTOLOGY AND PATHOLOGY

Structure and Formation of the Tubercle

Tubercule formation is evidence of a mechanical irritation, the presence of a foreign body in the tissue. The presence of the tubercle bacillus or particles of some inert foreign matter in the tissue may lead to the formation of tubercles but the chief cause of tubercle formation is the presence of the Koch's bacillus, its growth and multiplication, the chemical action of its metabolic changes, its toxin action as a foreign protein, etc. Tubercle formation is really nature's first effort at healing, showing a beginning immunization and a beginning chronicity of the tuberculous process. In all those instances in which resistance is low, virulence high and the soil propitious, no tubercle formation takes place. Tubercle formation is only present if the disorder shows a healing tendency (89). It is nature's efforts at arresting the tuberculous process, is evidence of body resistance. In acute rapidly progressing and fatal cases of pulmonary tuberculosis, tubercle formation generally does not take place, is entirely wanting.

Genesis of the Tubercle. v. Baumgarten's classic experiments on animals, by injecting live, virulent tubercle bacilli into the anterior chamber of the eyes of rabbits, has clearly demonstrated the growth and development of the tubercle from its inception to its completion as a fully developed body. His technic was as follows:

A number of rabbits treated in a similar manner were killed at various intervals and the enucleated eyes carefully studied; resulting in the following observations. After the first few days following the implantation of bacilli into the anterior chamber of the eye, nothing is noticeable except a slight increase in the number, gradually invading the contiguous structure of the iris and cornea. About the 6th day, distinct histologic changes at the point of entry are noticeable and in the regions where the bacilli are most numerous cell proliferation and newly developed epithelioid cells from connective tissue cells are observed. As the

tubercle bacilli increase, the epithelioid cells increase correspondingly, and about the 7th day indirect cell division or karyokinesis is noticeable in the fixed cells. This infiltration increases in extent and now endothelial, and connective tissue cells take part in the process, at this stage many bacilli within and without the epithelioid cells in the tubercle may be seen and now leucocytes also seems to take part in this process, and many bacilli are found within their bodies. In this way the formation of the tubercles spread and by coalescence may form a conglomerate mass. Pre-

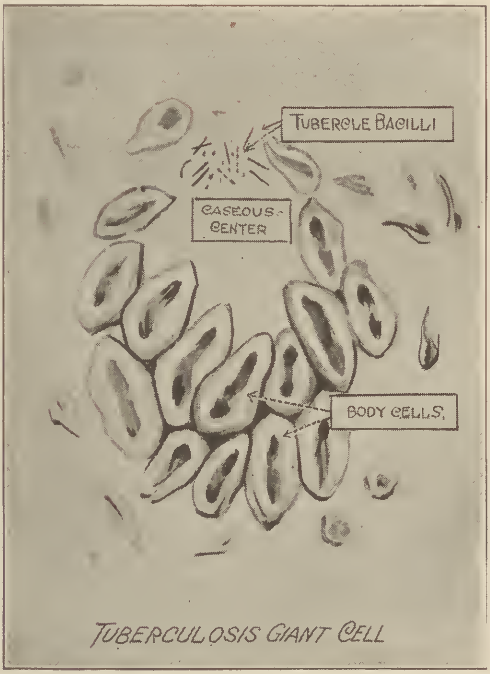

Fig. 7. Tuberculosis Giant Cell. (From Koch's original paper 1882). Note the tubercle bacilli at one pole and the body cells (nuclei) at the other. (H. J. Corper Lectures on Tuberculosis. Bulletin Chicago Tuberculosis Institute, 1916.)

cisely the same process of tubercle formation takes place when bacilli enter any part of the human body.

The action of the tubercle bacillus on the surrounding tissue is found to be four-fold, namely, tissue proliferation with tubercle formation followed by exudative inflammation and necrosis or fibrosis. The tubercle as we see it consists of epithelioid, connective tissue and endothelial cells surrounding the bacilli. After a time cell division ceases and the bodies of isolated

epithelioid cells begin to enlarge and containing many nuclei (59); these are designated giant cells and within them few or many bacilli may be observed. If the process is chronic, as in scrofula or joint disease, only a few isolated bacilli are found, whereas if the process is acute, as in active pulmonary disease, the number of bacilli contained in a giant cell may be as many as 50 or more, and then again if the bacilli are few in number, many giant cells are usually present, but if the bacilli are many, only few giant cells may be observed. Another peculiarity of the giant cells toward the bacilli is the supposed antagonistic relationship existing between the nuclei of the cells and the contained bacilli. If the nuclei within the giant cell lie at the outer area, the bacilli will be found at the center, and if the nuclei are found heaped at one pole, the bacilli will be found at the opposite pole. In this now completed tubercle, between the giant and the epithelioid cells, many wandering cells, round cells and leucocytes with well staining nuclei are noticeable, the latter playing a most important part in the formation.

Throughout all is a fine network or reticulum of fibrous bands consisting of threads, fibro-blasts, spindle cells and fibro-blastic tissue from connective tissue cells. The regional vessels now relax, allowing leucocytes to pass freely through their walls. No blood vessels are found in the tubercle, the nourishment being supplied from the lymph spaces.

Encapsulation, Caseation or Softening. About the 10th or 11th day an inflammatory process is observed, and with the lessening of cell proliferation many leucocytes (lymphocytes) are seen passing through the capillary vessel walls into the deposited cells. Fibroblastic cells now predominate the whole structure. If many leucocytes now surround this formative process, the tubercle may remain of the small cell type and none or only an isolated giant cell be observed. The tubercle may then undergo further encapsulation and fibrosis or may degenerate or caseate. The tubercle is usually complete in about 14 days and the course that now follows depends upon many factors:[1] retrogression, coagulation, necrosis or softening usually results. The bacilli

[1]With the earliest formation of the tubercle, throughout its whole existence there is a constant attempt at either a progression or retrogression of the process, at death or repair, and while the tubercle may undergo degenerative changes at its centre repair may take place at the periphery, hence the fate of the tubercle is entirely dependent upon the tissue reaction. If degenerative changes with necrosis are in the lead then the process will be progressive and with more or less rapidly invading and spreading into contiguous tissue, on the other hand if fibrosis, which is equivalent to repair, controls the situation then retrogression, arrest or checking of the process and healing results.

may die, the tubercle may become sclerotic or it may calcify, caseate or undergo cheesy degeneration, may soften and be absorbed or may break down, slough be followed by pus formation, or many tubercles may coalesce, forming a conglomerate mass. The tubercle in its entirety forms a small gray transparent nodule, in size that of a millet seed containing many or few bacilli and no blood vessels. If caseation takes place, the tubercle bacilli in the center die, the tissue undergoes retrogressive changes and fatty degeneration, the leucocytic protoplasm disappears, the nuclei degenerate, and with the entrance of pus germs and the presence of sterile pus, the process is completed.

At this stage the nuclei have lost their staining quality and a more or less cellular debris is present. The caseation, softening and pus formation is not due to the ordinary specific pus cocci, staphylococci and streptococci but most probably to a proteolytic ferment inherent in the tubercle bacillus itself analagous to the softening of a pneumonic exudate by the digestive ferments of the dissociated leucocytes.

The development of the tubercle may be divided into 5 phases:

1. A sero-fibrinous exudate following the implantation of the bacilli, brought about by mechanical irritation.

2. This mechanical irritation next brings about an aggregation of many leucocytes (Polymorphonuclear) which, however, die in a few days, fading away without leaving a trace.

3. Connective tissue cell reaction with formation of epithelioid cells, increase of nuclei and cell protoplasm with karyokinesis; endothelial, and fixed connective tissue cells are all changed into epithelioid cells, a reticulum is noticeable and phagocytosis is demonstrable.

4. Now mononuclear leucocytes at first only isolated appear in great number in the periphery of the tubercle.

5. Degeneration of the tubercle and a second entrance of the Polymorphonuclear leucocytes.

In both the second and fifth phases the polynuclears play a leading role due to cell irritation and toxic causes. The leucocytes present in the first phase are due mainly to irritation.

Early, from his studies, Koch promulgated his views concerning the formation of the tubercle. He contended that the bacilli entering the body are taken up by the leucocytes, through the lymph and blood supply, and are deposited in the tissue. The leucocytes are first changed into epithelioid cells and later into

giant cells. The produced toxins change the surrounding cells into epithelioid cells, the tubercle ultimately undergoes necrotic changes or fibrosis and exudative processes from inflammatory surroundings join in the disorder. If the process assumes a curative form encapsulation by means of fibroblastic tissue formation takes place, a fine network permeating the whole tubercle, the caseating mass being absorbed or undergoing calcification.

In the tuberculous, at autopsy, histological changes are noticeable in all the various organs of the body and these are either typical tuberculous tissue or simply chronic inflammatory changes and not tuberculous lesions proper.

The Usual Location of the Tubercle and Its Ultimate Fate. In most instances the beginning tuberculous process is in the terminal bronchioles, a bronchiolitis tuberculosa resulting. At the point where the cylindrical epithelium (cuboidal cells) of the bronchioles joins the (non-nucleated Polygonal cells) alveolar pavement epithelium, it is just beyond the ring of muscle fibres in the terminal bronchi described in Chapter 7; the alveolar septal walls and alveolar sacs are also with like frequency involved. The most favorable selection is along the right posterior bronchus, the Bronchus Apicalis Posterioris and including the interstitial connective tissue a peribronchiolitis is produced. If only a single nodule, it may be early encapsulated, then undergo calcarious degeneration and in time be expelled as a hard mass, (the so-called lung stones, pneumoliths) leaving a contracted scar; it may undergo cheesy degeneration, the bacilli may be scattered and many metastatic nodules appear, or again many nodules may conglomerate, degenerate, break down and lead to cavity formation.

CHAPTER 9

IMMUNITY

General Consideration. The word immunity is derived from "Immunis" free from paying tribute (19). It simply expresses an idea, no well defined conception. Immunity the freedom from becoming sick upon the entrance of bacteria or their toxins into the human or animal body and the possibility of certain micro-organisms or their toxic products entering the body and making it sick or causing death is known as virulence. If, however, the human body resists the invasion of these various bacteria and thereby prevents the bringing about of disease, then we speak of the body's defense forces or immunizing agencies and the forces necessary are known as the immunizing mechanism. On the other hand, if the body is passive, permits the invasion and does not resist implantation, then we speak of virulence of the invaders; hence we notice that immunity and virulence are strictly antagonistic forces.

In many instances immunity depends entirely upon the degree of virulence and the amount of disease producing germs entering the body; it also depends a great deal upon the individual organism. For instance, a streptococcus strain may be highly virulent to a certain individual and cause death; the same strain in another individual may not even produce any symptoms of disease; it may be avirulent to one and highly virulent to another. If the body is capable of arousing the necessary defense powers when micro-organisms enter, the invaders become avirulent and will produce no disturbance; if, however, the body possesses no such defense powers, or perhaps the resistence is lowered by either an acquired or inherited disposition, then the entering bacteria will grow, multiply and eventually cause disease or even death. Again, the same individual at times may be fully capable of resisting an invasion whilst at other times he may possess no power whatsoever to prevent it.

In practical medicine it has long been recognized that fatigue, injury, nervous shock, psychic influences, extremes of cold or heat, etc., play leading parts in body resistence. It will be noticed

that the invading micro-organisms and the resistance offered by the body cells is very similar to an invading army of an enemy and the resistance offered by the defenders. This simile may be observed in more than one way.

It becomes apparent from this that many micro-organisms possess more or less, an infection possibility depending on either a heightened resistance or a lowered vitality, a susceptibility or a non-susceptibility of the body at the time of infection. However, if the mechanism of immunity is functioning well, there will be no susceptibility; if not, the body is susceptible to infection, and to disease and death as well. Usually with the entrance of the bacteria into the organism, immunizing activities are brought about and directed against the invaders, and conversely, when micro-organisms remain for some time in the body, they become immunized against the body's defense powers. Immunity and virulence as well are but relative terms; there exists no absolute immunity; only a partial immunity can be achieved. Under certain conditions immunity always exists and under certain other conditions it can always be lowered, depending upon whether or not at the time the immunity of a given individual is sufficient to be utilized for practical purposes.

Immunity may be congenital or it may be acquired later in life. A **congenital immunity** is present in some individuals without having previously come in contact with the disease producing germs. Such individuals when coming in contact with a pathogenic virus give no manifestation of either infection or disease. This is a fixed immunity and can not be transmitted to other or non-protected beings; not so the acquired immunity. This is added during life to the non-immune body as a new function, is always specific, always for the same organism and no other, and may be either active or passive.

The Acquired Tuberculous Immunity (123). It is now a well established fact that most civilized people during some time in their lives have become tuberculously infected and this leads us to recognize a second fact, namely, that in but a very small percentage is this infection followed by disease.

We are all familiar with the fact that the application of tuberculin is, in most individuals, followed by a positive reaction; however, this is no evidence of positive disease, it only proves that the reacting person at some time in his life came in contact with the tuberculosis virus, but should we conclude from this

that all who react to the tuberculin test, and show no signs of clinical manifestations are tuberculosis immune?

Throughout our whole life, particularly in the large cities, we come constantly in touch with the tubercle bacillus, through our daily intercourse with one another, but these later infections are made perfectly negligible from the acquired immunity incident to a previous or earlier infection, our bodies showing an ever increasing defense, increasing more and more with every new infection, in this way increasing the immunity which with the defense products go hand in hand. It is not necessary that the established immunity destroy all the tubercle bacilli in the organism; it is only necessary to keep the infecting virus constantly in check. This so constantly renewed and reacquired immunity protects the body from infection from without and from within as well.[1]

That recovery from a tuberculous infection offers a high degree of immunity was positively established by v. Behring in his epoch making experiments on cattle, in immunizing against bovine tuberculosis by intravenous injection with living human tubercle bacilli, and Koch's early contention that a tuberculous infection in animals protects them from a subsequent reinfection has been repeatedly verified.

In the human after the infection has passed off, the organism is again free to infection and although an immunity may continue for some length of time to a high degree, it gradually lessens, and now a recurring or repeated infection again increases this immunity and so the power continues throughout the great part of our lives, with a constant increase and diminution of immunity.

About three-fourths of all human beings have come and are constantly coming in contact with minute quantities of tubercle bacilli and in this way are continually maintaining their immunity. If, however, this reinfection with the virus should be in too great an amount or if the virulence produced is very large, the body may not be capable of supplying the necessary defense products and so can not sustain its previously acquired immunity, the infection then is soon followed by manifest tuberculous disease. The human organism which has survived a tuberculous infection is in its behavior quite different from one which has never come in contact with the virus. Because of this

[1]Although the recovery from a tuberculous infection in the ordinary way will give relatively an immunity we have not succeeded as yet by artificial means therapeutically to bring about a specific immunity against infection.

so general infection in the human, most children born possess from birth a slight degree of tuberculous immunity and are from infancy already able to overcome a slight infection, not so the people and their children who have never come in contact with the virus. We frequently observe in primitive races that a primary infection even if this infection is very slight is often followed by most disasterous consequences, a rapid tuberculous process, and this is because they possess neither an inherited nor an acquired immunity. This shows that immunity is only ac-

Fig. 8. The relative death rate in the U. S. among Indians, Colored and Whites (comparatively). (From the U. S. Public Health and Marine Hospital Service. Tuberculosis exhibit.) (Chicago Tuberculosis Institute Film.)

quired by years of contact with the virus. For instance, we observe that the white race is much more resistant, is much more immunized to a tuberculous infection and disease than is the colored and the colored again more than the American Indian. This is most natural when we consider that the white race has come in contact with the tubercle bacillus for many thousands of years whilst the colored race began its immunization only since its association with the white many hundreds of years ago, and the Indian since the discovery of this country, some four hundred years.[2]

[2]A frequently repeated but mild tuberculous infection constantly adds to the small capital of an inherited immunity which was given to the infant at birth. With each new mild infection this capital of immunity increases so that in years after the infected individual can utilize the interest accrued on this capital in the form of resistance to protect him against a tuberculous disease.

A tuberculous process which has been healed for some length of time (say for years) has left no protecting immunity and hence it leaves the individual liable to reinfection; it is only in the tuberculous organism that immunity is present. Only the tuberculously infected organism is capable of immunizing against this disease, and not the non-infected. It is this constant reinfection from without which takes place in our daily life which gives us immunity.

It is most probable that bacterial toxins, which are necessary to bring about immunity, are produced in the living body in response to the stimulus of the protective agencies secreted by the body cells. In both bacteria and body cells there exists a constant attempt at immunization, bacteria trying to make themselves immune against the body cells and the body cells in turn endeavor to immunize against the bacteria.

On a previous page mention has been made that Ghon in his 184 autopsies found in by far the greater number (142) but a single primary focus from which he concludes that this first inoculation stimulated the production of sufficient defense agencies to give immunity against a later or second infection. Not only in the experimental animal can immunity be instituted but in the human as well do we find an acquired, an autogenous immunity. This autoimmunization is brought about by the stimulating activities incident to the tuberculous infection, and this now gives to the body a relatively protecting immunity.

The discovery of healed lesions or healing foci in about 90% of all autopsies and the fact that in only about 10% do we find tuberculous disease the cause of death shows that the greater majority of all human beings possess in some degree an acquired immunity against this disease.

The various methods of tuberculous immunization. Many attempts at immunization in both man and animal by means of the human tubercle bacillus, either living or dead, by the bovine, the avian, the cold blooded animal bacillus, the various acid fast bacilli of this group as well as by the use of the artificially produced toxins have been made. Many of this group have been used, tried and experimented with ever since the discovery of the tubercle bacillus, and from much of this experimental work we have learned that the highest degree of immunity for a time, not absolute, can best be secured by inoculating the organism with the living tubercle bacillus which possess a superiority over

the dead bacillus and all the recognized bacillary products for a protective inoculation. Usually only a minute quantity or a single bacillus is used, in the beginning, this dosage being gradually increased until the experimental organism becomes tolerant to extremely large doses. It is supposed that the use of the various tuberculins for immunizing purposes are equally as efficient as the living bacteria, perhaps in a somewhat less degree they are at the same time less dangerous. By the use of these various agencies varying degrees of immunization can be achieved, the method of subcutaneous or intravenous injection being followed by the best results. By the administration of the bacilli by mouth, the feeding method, good results are also obtainable, and a noticeable resistance in animals to an artificial tuberculous infection may be brought about by feeding cattle on emulsion of living bovine tubercle bacilli, either virulent or modified by heating to 70°. A single ingestion may be sufficient to produce an infection, and subsequent immunization, the animal being capable of resisting for many months a dose sufficient to infect a control animal; however, we must bear in mind that by the feeding method much larger doses must be used and that an initial dose of the tubercle bacillus by way of the digestive tract is about 6000 times that of the number inhaled.

If to an experimental animal treated as above we give an intravenous inoculation of a 5 milligram dose of virulent bovine bacilli and a similar dose be given to a control animal of the same age, the control will develop an exceedingly rapid tuberculosis which will prove fatal within 6 weeks, while the previously treated animal will remain perfectly healthy for about 8 months, and then lose its immunity, developing a sudden and rapidly fatal tuberculosis.

Animals previously tested with tuberculin are very similar in their behavior to those inoculated with the living virus, and intravenous injections of tuberculin will give to an animal an immunity not possessed by controls. If 5 milligrams of virulent bovine bacilli are injected into an animal a few days after the tuberculin injection and the same amount be given to another animal as control, it will be found that the control will perish in about 6 weeks while the previously prepared animal will present a slowly progressing process. There obtains no doubt that both tuberculously infected and healthy animals, if previously treated

with large doses of tuberculin, possess incomparably greater resistance than animals not so treated.

A local suppurative tuberculous process occurring in a consumptive often improves the individual's general health and greatly increases his resistance, raises his immunity, and conversely, a person in whom pulmonary tuberculosis is running a rapid course rarely gives a history of a previous suppuration either of a gland, bone or any other form of local tuberculosis. This local disease has stimulated the defense powers to a relative immunization, and as long as this can be maintained, the individual enjoys comparatively good health. Thus the history which we physicians so often hear in our daily routine work especially from elderly people, that a long standing and discharging ulcer on the leg or any part of the body should not be healed because if cured it is often quickly followed by most disastrous consequences, (perhaps by active pulmonary tuberculosis) may not be without foundation. May we not here also consider the belief of the ancients in the insistence of a pre-existing catarrh constituting an open door to a tuberculous disease? A previously existing tuberculosis or a tuberculous lesion in process of evolution gives a relative protection or immunity and prevents the evolution of a new or second inoculation.[3]

Hypersensitiveness, Sensitization, Etc.

If the healthy organism receives a single inoculation with sera or bacteria, the body becomes very much sensitized, even if the second injection of a similar substance has been given in very much smaller dosage. This phenomenon is known as "the reaction." The cause of this hypersensitiveness is due to definite bodies in the blood serum, the result of the primary inoculation, which are known as lysins. If the invaders are in small number, they are destroyed by the lysins, and this destruction is followed by the reaction; if, however, the bacteria are in great numbers, then by means of the lysins, solutions of the invaders are brought about, and this may lead to most serious intoxication.

In a sense hypersensitiveness and immunity are practically interdependent, there exists between them a causal relation. The body's sensitiveness to the application of tuberculin is evidence of the body's resistance; it shows that antibodies or protective

[3]The extreme frequency of a positive tuberculin reaction, on the one hand, and the evidence of completely healed foci at autopsy on the other, goes to prove that already in the child tuberculosis is relatively a harmless disease and that tuberculosis is very frequently only a latent process never manifesting any symptoms.

agencies are at work at the time. Sensitiveness to vaccines or to bacillary products is spoken of as anaphylaxis or as an anaphylactic reaction, or more correctly as allergy and the absence or want of this sensitiveness, which we may observe frequently in those who never come in contact with the virus, is known as anergy. (See Chapter XXXX.)

The various tuberculins, and they are very numerous, are all being used to bring about active immunization in the infected and also the sera, Marmoreck's, Maragliano's, etc., with which only a passive immunization is aimed at. It is, however, very doubtful if in tuberculosis passive immunization is of any value. An activo-passive immunization is sought by the use of Carl Spengler's I. K. or immune bodies (Immune Koerper) but it is very questionable if any degree of immunity can be established with any certainty in this way.

PART TWO

CLINICAL TUBERCULOSIS

Having first considered the various academic questions concerning tuberculous disease we are then in a position to continue the subject from the clinical standpoint. Beginning, somewhat in detail, the study of the symptomatology, then the diagnosis, prognosis and treatment of pulmonary tuberculosis, as observed in the adult we shall continue to consider this disorder as it is observed in children. The consideration of Tuberculin becomes our next lesson. Here many of the intricate topics and questions as observed are really of an academic nature and for that reason may seem to belong more appropriately to that classification, to part one, however, Tuberculin, is so closely and intimately connected with the diagnosis and the treatment of tuberculosis in all of its various manifestations that it appeared to me better to consider it on the clinical side rather than on the academic.

Some of the chapters on tuberculosis of other organs than those of the lungs or where pulmonary tuberculosis was complicated by tuberculous disease of other organs or tissues were compiled from lectures delivered and clinical instruction given to my classes, at different times during the school years by members of the teaching staff of Rush Medical College.

CHAPTER 10

PULMONARY TUBERCULOSIS. PHTHISIS PULMONUM. PHTHISIS PULMONALIS. PHTHISIS. CONSUMPTION, TUBERCULOSIS, ETC. SCHEMATIC CLASSIFICATION.

Under the generally known generic term "Tuberculosis," we usually designate a diseased condition of some organ or tissue of the body, brought about by the presence of the tubercle bacillus, and the name of the individual organ which is involved as the specific term: Phthisis (generic) Pulmonalis (specific). Every tissue and every organ of the human body may be the seat of the bacillary invasion, but in by far the greater number of instances we observe, especially in adult life, that these invaders have a decided predilection for the lungs. Besides the lungs, glands and perhaps the abdominal organs which are usually the seat of primary disease, all other organs become secondarily involved and only extremely seldom do we find these organs the seat of primary disease.

The pulmonary form of this disease was early recognized by the human family and because of the slow, protracted and wasting course it so frequently persues, it was called consumption.

Ever since the discovery of the tubercle bacillus as the causative factor, this disease has been most intensely and thoroughly studied, and from the knowledge thus acquired rules have been arbitrarily formulated for our guidance which may enable us more readily to recognize the disorder.

Pulmonary Tuberculosis is a progressive disease. In many instances the disease begins as a very slight infiltrative process extending slowly, as there is no pain, there is usually absence of all subjective symptoms and in this way the disease has often spread and extended into neighboring tissue before the physician is consulted. In other instances, the disease runs a more rapid course, is more rapid in its process of invasion and produces more recognizable symptoms, both subjective and objective. Individuals affected with this form of the disease are more free in seeking medical advice. There is still another class, known as the miliary

in which the disease runs a more or less acute form and is generally quickly fatal.

The duration of pulmonary tuberculosis is quite variable. It may from the beginning manifest a very stormy scene, running a rapid course and exitus letalis in a few months, or it may be much protracted, running a slow course lasting for years. Many chronic cases are known to have lasted from 10 to 20 or more years; however, it can be stated that the average duration of pulmonary tuberculosis is about two years.

Various attempts at classification of patients on examination have from time to time been made so as to recognize, from the amount of lung involvement present, to which class or group a certain individual belongs. Such an attempt at classification we already find in the pulmonary histories of the ancients, where mention is made of Phthisis Incipiens or beginning tuberculosis, Phthisis Confirmata or established phthisis, conforming to our now so much used term moderately advanced phthisis and Phthisis Desperata or grave phthisis, which we now recognize as the far advanced type.

The committee on nomenclature appointed by the National Tuberculosis Association some years ago formulated a classification based on the Turban-Gerhardt Scale which is now generally accepted. This classification (the Turban-Gerhardt Schema) is as follows:

National Tuberculosis Association Schema for the Classification of Patients on Examination

The following definitions indicate the farthest extent of disease and the greatest severity of symptoms that a patient can present and still belong to the stage defined. All patients beyond the incipient stage (first stage) fall under the moderately advanced stage (second stage) unless the physical signs and the symptoms exceed those of the moderately advanced stage, when they should be classified as far advanced (third stage).

Incipient—First Stage

Slight or no constitutional symptoms (including particularly gastric or intestinal disturbance, or rapid loss of weight).

Slight or no elevation of temperature or acceleration of pulse at any time during the twenty-four hours.

Expectoration usually small in amount or absent. Tubercle bacilli may be present or absent.

Slight infiltration limited to the apex of one or both lungs, or a small part of one lobe.

No tuberculous complications.

Moderately Advanced—Second Stage

No marked impairment of function, either local or constitutional.

Marked infiltration more extensive than under incipient, with little or no evidence of cavity formation.

No serious tuberculous complications.

Far Advanced—Third Stage

Marked impairment of function, local and constitutional.

Extensive localized infiltration or consolidation in one or more lobes.

Or disseminated areas of cavity formation.

Or serious tuberculous complications.

Acute Miliary Tuberculosis—Generalized

In this classification, each of the three stages is based on certain specific pathological findings and clinical symptoms. Unfortunately, however, lesions and symptoms are not interchangeable, but are immobilized in each of the three stages. This is confusing, for occasionally an advanced case may present only incipient symptoms, or vice versa.

The Turban-Gerhardt Schema

FOR THE CLASSIFICATION OF PATIENTS ON EXAMINATION

I. Slight lesion extending at most to the volume of one lobe or two half lobes.

II. Slight lesion extending further than I, but at most to the volume of two lobes; or severe lesion extending at most to the volume of one lobe.

III. All lesions which in extent of the parts affected exceed II.

By "slight lesion" we understand disseminated centres of disease which manifest themselves physically by slight dulness, by harsh, feeble, or broncho-vesicular breathing, and by rales.

By "severe lesion" we mean cases of consolidation and excavation such as betray themselves by marked dulness, by tympanitic sounds, by very feeble broncho-vesicular, bronchial, or amphoric breathing, by rales of various kinds.

Purely pleuritic dulness, unless marked, is to be left out of account; if it is serous, the pleurisy must be specially mentioned under the head of "tuberculous complications."

The volume of a single lobe is always regarded as equivalent to the volume of two half lobes, etc.

A supplemental report

"At the meeting of the Committee in New York City, on December 12th, 1915, a rearrangement of the classification adopted by the National Tuberculosis Association was presented to the members for consideration. Owing, however, to the limited time at their disposal, a thorough discussion on the subject was not possible, and the President, therefore, appointed a Committee of three members with instructions to submit a report on the proposed rearrangement, at this meeting. The Committee realizes that frequent changing of the classification is inadvisable, and appreciates the necessity of maintaining a standard schema. It is also aware of the confusion that may arise should the text of the present classification be materially modified, and realizes that a radical change would seriously affect the past statistics of the various sanatoria. The Committee has, therefore, confined its proposals to a simple rearrangement, which will increase the flexibility of the classification, without in any way altering its text. The members of the Committee feel that, owing to the wide variation in symptomatology, which is frequently manifested by patients in the same stage of the disease, a rearrangement of the classification is advisable, and it is not possible to immobilize lesions and symptoms in the same stage in any schema, without decreasing its range of application. The present classification describes the lesions and symptoms presented by the general run of cases admirably, but as it confines both the lesions and symptoms to separate stages, it fails to classify satisfactorily many cases of pulmonary tuberculosis. The proposed rearrangement divides the lesions and symptoms of the schema into separate groups and lists them under different headings. It offers nine combinations and makes possible the use of any of the three groups of symptoms with the lesion of any stage. It covers the borderline cases so frequently found in the moderately advanced class and likewise provides for the classification of all cases within the scope of the present schema.

The present rearrangement, which contains the same terms used in the former schema, with the exception of those enclosed in parentheses, follows:

LESION	SYMPTOMS
A. **Incipient.** Slight infiltration limited to the apex of one or both lungs, or a small part of one lobe. No tuberculous complications.	A. (Slight or none.) Slight or no constitutional symptoms including particularly gastric or intestinal disturbance, or rapid loss of weight; slight or no elevation of temperature or acceleration of pulse at any time during the twenty-four hours. Expectoration usually small in amount, or absent. Tubercle bacilli may be present or absent.
B. **Moderately Advanced.** Marked infiltration more extensive than under incipient, with little or no evidence of cavity formation. No serious tuberculous complications.	B. (Moderate.) No marked impairment of function, either local or constitutional.
C. **Far Advanced.** Extensive localized infiltration or consolidation in one or more lobes. Or disseminated areas of cavity formation. Or serious tuberculous complications.	C. (Severe.) Marked impairment of function, local and constitutional.
D. **Acute (Generalized) Miliary Tuberculosis.**	

This schema offers the following combinations:

Incipient A	Moderately Advanced A	Far Advanced A
Incipient B	Moderately Advanced B	Far Advanced B
Incipient C	Moderately Advanced C	Far Advanced C

Incipient A, depicts an Incipient lesion with symptoms as found in the present Incipient Stage; Incipient B, an Incipient lesion with symptoms of the present Moderately Advanced Stage; Incipient C, an Incipient lesion with symptoms of the present Far Advanced Stage; Moderately Advanced A, a Moderately Advanced lesion with symptoms of the present Incipient Stage; Moderately Advanced B, a Moderately Advanced lesion with symptoms of the present Moderately Advanced stage, etc.

It is recognized that an Incipient C case is a rarity, but, nevertheless, the condition does occur and should be covered by the classification. Incipient B cases occur more frequently. Moderately Advanced A cases are common, and those in the Moderately Advanced C class are not frequently encountered. Patients of the Far advanced B and Far Advanced

A classes are abundant in hospitals for the treatment of advanced cases. The rearrangement gives an accurate idea, at a glance, of the amount of lung involvement and the symptomatic picture. Much has been said in the literature of late in regard to the occurrence of localized pulmonary miliary involvement, and it might be advisable to classify "Acute Miliary Tuberculosis" as "Acute Generalized Miliary Tuberculosis".

A classification which I have taught for years is based on this National one, but it is slightly modified for somewhat easier comprehension by students. It is as follows: A tuberculous involvement, if situated in both upper apices above the clavicles or posterior above the spine of the scapulae, may be spoken of as a first stage case; if the involvement is on one side only and it has not extended below the second rib, then it is also said to be a first stage case. Posteriorly, the involvement in such a case would extend slightly below the spine of the scapula, most probably as far as the second dorsal spine. Now, as the disease progresses with more or less rapidity into contiguous tissue and at the same time downward, we usually find that where one apex has been primarily involved, for example, say the right, the apex on the opposite side, the upper, soon becomes the seat of secondary disease, and simultaneously with this the apex of the lower lobe on the side originally involved (the right). In exceptional instances this lower apex is not the seat of secondary disease, but by crossed infection both the upper and lower lobe of the opposite side become secondarily involved. This involvement of the lungs, that is the apex of one side primarily and the apex of the lower lobe of the same or the opposite side and the upper apex on the opposite side, if found in an individual, can be said to be a second stage case, and all involvement beyond this, that is, involvement of all the apices of the lungs as well as a greater portion of the body, if present, can be diagnosed as a third stage case. Tuberculosis known as the Generalized Miliary Form is considered separately on page 96.

A classification of clinical symptoms and physical signs in Tuberculosis as outlined by F. M. Pottenger may be of interest to students and is given herewith. It offers no special advantage over the grouping into the prodromal or preactive and the subjective and objective or active symptoms as first given by me and clearly outlined in the text. (See Chapter 11.) The classification which he offers is as follows:

(1) Those due to toxaemia, which fully correspond to the prodromal signs due to intoxication as mentioned in my classifi-

cation: (2) Those due to reflex action and (3) Those due to the tuberculous disease itself. The latter two correspond to the signs and symptoms of active disease in tuberculosis.

Group 1. Toxaemia. Malaise, lack of endurance, loss of strength, appetite and weight, nervous and digestive disturbances, anemia, rapid pulse, temperature and night sweats.

Group 2. Symptoms of Reflex Signs. Hoarseness, cough, irritation of the larynx, digestive, circulatory and vaso-motor disturbances, chest and muscle pain, loss in weight, anemia.

Group 3. Peculiar to the disease. Frequent and protracted colds, hemorrhages, expectorations, pleurisies, pulse and temperature, etc.

The National Tuberculosis Association gives the following definition of incipient or beginning tuberculosis:

Slight or no constitutional symptoms (including particularly gastro-intestinal disturbances, or rapid loss of weight).

Slight or no elevation of temperature or acceleration of the pulse at any time during the 24 hours.

Expectoration is usually small in amount or is absent. Tubercle bacilli may or may not be present.

Slight infiltration limited to the apex of one or both lungs or a small part of one lobe.

No tuberculosis complications.

This definition indicates the farthest extent of lesion or disease possible to be classified as incipient tuberculosis, and corresponds closely with the Turban-Gerhardt scale—(Turban 1) "a slight lesion extending at most to the volume of one lobe or two half lobes."

Is it not evident after scanning the above classification that so-called incipient pulmonary tuberculosis is now no longer a beginning disease, that with such findings the stage of incipiency how long since passed, and is now a fully established disorder, particularly when we see it plainly stated that bacilli may or may not be present? The word "incipient" denotes the beginning of something. Accepting this definition, the term incipient tuberculosis as applied above should be either eliminated or used in a more restricted sense, because the disease is now no longer in the beginning stage. If the conditions of

incipiency, as above outlined, have existed for months or perhaps years, all depending upon the extent of involvement and the rapidity of the pathological process, is it then logical or reasonable to call the stage incipient when on repeated examinations we find the same physical signs? In the history of no other disease do we continually speak of a beginning disorder when we have before us all the evidence of a long existing malady.

Primary or established pulmonary disease (144) should be designated by such descriptive terms as "early tuberculosis," as "manifest tuberculosis" or as "first stage disorder," and the expression "incipient tuberculosis" used to denote a definite beginning involvement only. In speaking of early established pulmonary disease such descriptive terms as mentioned, no matter how long the disease has existed or how much primary involvement may be present, are more correct, appropriate and less misleading than the term "incipient" now in use.

Pulmonary tuberculosis generally after being manifested for some time gives evidence of body disturbances, and such disturbances are accompanied by symptoms which are either subjective or objective, that is, such as are observed by the tuberculous person and such as are noticed by the examining physician, however, we shall first consider such signs as are frequently present, but are usually not observed (for a long time), that is, before the disease becomes evident, and which are designated as Prodromal "Signs and Symptoms".

CHAPTER 11

THE SYMPTOMATOLOGY OF TUBERCULOUS DISEASE

The symptoms of activity in pulmonary tuberculosis are of the greatest importance, even more so than are the physical signs. Clinical symptoms if present in a given case leave no doubt as to a diagnosis, whereas physical signs alone may still give doubtful findings.

Pulmonary tuberculosis, as stated, is a quantity infection, hence the number of bacilli that have been deposited in the lung tissue within a given time is of primary importance. If only a few bacilli or only a single tubercle undergoing degeneration is present and the extension of the process is slow, then the recognizable symptoms of tuberculosis may not manifest themselves in months or even years, whereas if the infection is massive, that is, if many bacilli enter the pulmonary tissue at once, producing many individual colonies and many separate tubercles, and because of more separate colonies produce more toxicity, and if these tubercles now break down rapidly, causing much destruction of tissue, then the accompanying symptoms are pronounced, and as is the case in miliary tuberculosis, become hematogenous, overwhelm the system and cause a speedy death. In no other disease are the symptoms so protean. They may be mild and insignificant, not noticeable by the patient nor suspected by the physician, drawn out over long periods of time, over months and even years, with only a slight indisposition now and then, whilst on the other hand the symptoms may be so pronounced and overwhelming as to baffle both the patient and the physician. When the disease has been once definitely established, the symptoms are observed as subjective and objective. Subjective such as are noticed by the infected individual, and objective, those recognized by the examining physicians. Long before the disease is clinically established in the lungs toxic symptoms may be observed, but only too often they are supposed not to relate to pulmonary disease. These symptoms are known as the prodromal or premonitory. Symptoms may be divided into two groups: those of latency and those of activity.

1. **The symptoms of latency**; the symptoms of preactivity, the prodromal symptoms; symptoms of toxicity, the symptoms of localization, the warning symptoms; the symptoms of the first stage or the stage of latency; the stage of intoxication; the early signs and symptoms of tuberculosis.

2. **The symptoms of activity**; subjective and objective. The symptoms of bacillary growth and development; the symptoms of dissemination and spreading of the tuberculous process; the symptoms of the stage of active pulmonary disease; the second stage, the stage of activity, of manifest disease, including the signs and symptoms of early tuberculosis.

A. Prodromal Symptoms—Symptoms of Latency

In the tuberculous individual that is early in the disturbance when the disease is still localized, perhaps confined to a single or at least to only a few tubercles, only few symptoms may be noticeable. The causes of these latent symptoms are due mainly to the very slight absorption of the elaborated toxins, the products of bacillary metabolism, and to the presence of foreign particles in the pulmonary tissue, giving evidence of irritation and inflammation, producing the variable picture of auto-infection. These symptoms are variously designated as *latent, occult, insidious, hidden, larval, obscure, subclinical, pretuberculous, etc.* The prodromal symptoms in tuberculosis are as positive and distinct as are the prodromal ones in the acute diseases, and they are always present. They differ only in being protracted or long drawn out, covering long periods of time, in many instances months and even years.

The symptoms of intoxication are: 1. Malaise,[1] a tired feeling

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[1] Is not the very tired feeling so often observed in beginning tuberculous disease in reality a sign of "conservatism" to compel the patient to lie or sit down and rest? We know that many cases of rapidly progressing active disease have almost never shown prodromal symptoms, the disease having been ushered in fully developed with all the signs of protracted collapse which we so frequently find in terminal cases. From the very beginning of the tuberculous infection and showing a tendency to become active, the organism assumes or nature asserts through the body a conservative attitude, and at all times nature is trying to check the invasion. It sends out an alarm that the hidden foe is coming forth and that the body must give battle. The very early, the prodromal symptoms, are more or less signs of warning and invitation for preparedness. Early signs are always present if the body shows the least resistance. These signs are only the noticeable evidence that the body is aware of the presence of the foe and is preparing to give battle. In most cases, although the conflict during the course of the disease is noticeable even if the disease is well established, still nature's efforts at conservation and resistance are always manifested. That these early or prodromal symptoms are really nature's alarm signs is evident from the observations that in rapidly active cases no such symptoms are manifest, nor are tubercles found in the diseased tissues, which again is evidence that the body offered no resistance at the proper time and that now the organism is overwhelmed with toxins. Immobilization and retraction are both signs of conservation and resistance, nature's effort to put the parts at rest while the body gains sufficient power to cope with the invaders, for if there is no rest of the invaded organs or at least that part of the organ which is actively concerned in the inflammatory process, then the disorder extends rapidly in all directions, leading to a quickly fatal course.

while at work, at play, taking a walk or on the least exertion; 2. Sleeplessness, distressing and painful dreams or disturbed sleep, particularly noticeable in young individuals at the school age, and are then attributable to overstudy; 3. nervous disturbances of various kinds, particularly more or less painful and protracted headaches lasting on and off for months or even years. Many cases, referred to as anemia, neurasthenia, hysteria, lovesickness, etc., are included in this category. 4. Cardiac disturbances, most noticeable in dizzy spells, shortness of breath on the least exertion, or on going up a short flight of stairs, and are usually referred to as heart disease, heart weakness or cardiac palpitation. 5. Gastro-intestinal disturbances, dyspepsia, nausea, vomiting, gaseous eructations, loss of appetite, hypo-acidity, all without any assignable cause, belong in this group. 6. Vasomotor disturbances, such as cold and clammy hands, persistent sweating of the hands or feet, free axillary perspiration, alternate paling and blushing of the face; if extending over long periods, these symptoms are in many instances directly attributable to intoxication. 7. Menstrual disturbances, dysmenorrhoea, menorrhagia, and particularly amenorrhoea have long been recognized by both the profession and the laity as probable forerunners of tuberculosis. 8. Rheumatism is often a symptom complex of a tuberculous intoxication. There are shoulder pains and usually one or more joints are swollen, reddened, painful and immobile with accompanying signs of anemia, emaciation, malaise, sweating. 9. Thyroid disturbances. The thyroid may be enlarged or may even simulate Grave's disease with slight tachycardia, palpitation, nervousness, sleeplessness, emaciation, etc. 10. Urinary symptoms.(73) An orthostatic albuminuria, but a complete absence of all cellular elements, the toxines producing a kidney permeability, this is known as a pretuberculous albuminuria.

That these are prodromal symptoms and are of tuberculotoxic origin can be demonstrated in various ways. 1. By the tuberculin reaction test. Individuals giving evidence of these symptoms usually react quickly and vigorously to an intradermal tuberculin test. 2. By relieving the symptoms. If to individuals manifesting these symptoms small or minute doses of tuberculin be administered subcutaneously over long periods of time, amelioration of all symptoms results. 3. By simulating these symptoms. If to individuals free from symptoms small or minute doses of artificial tuberculin be given hypodermically, all the

symptoms of a natural intoxication can be produced. This latter has repeatedly been demonstrated on children in whom a tuberculin test has been applied, for diagnostic purposes, when all the symptoms of a typical tuberculous rheumatism may manifest themselves.[2]

B. Subjective and Objective Symptoms. Symptoms of Activity.

These, in this the second stage or the stage of activity, are becoming more and more distinct and definite, the signs of early intoxication appearing now in the background and those of active disease manifesting themselves. Owing to the great chronicity of tuberculosis, the symptoms both subjective and objective continually change with the changed condition in the lungs, and the processes of latency, of activity, of quiescence, of reactivity, or of arrest may vary greatly, presenting a constantly changing clinical picture.

The usually pronounced symptoms of activity in the order of their importance and frequency, are as follows:

1. **Cough.** This may at first be slight or entirely absent, becoming more intense and distressing as the disease advances. The cough may at first be dry, or non-productive, assuming later a distinct moist character, or it may be distinctly brassy, even laryngeal, particularly if the disease has assumed a sudden exacerbation or rapidity. The cough may be painful and particularly productive during the night, with moderate expectoration during the day; this order may in isolated cases be reversed. The cough may be increased by lying on the unaffected side, or again the cough may be increased on taking food, especially after breakfast, which is often followed by vomiting. If the parenchyma of the lung only is involved, cough may be entirely absent, but is usually always present if the disease is peribronchial.

2. **Expectoration.** This may be very scanty or profuse; it may be scanty or wholly absent at the beginning or consist of a stringy mucous, later muco-purulent, more or less mixed with particles of dust, coal, fibres and fabrics, etc., very rarely bloody. If a long, protracted and painful cough is present, blood streaked sputa may be observed; later, as the expectoration increases, it assumes a more or less purulent or yellowish-gray appearance. In the very chronic forms of the disease, there may be very little

[2]These prodromal symptoms or disturbances may manifest themselves in the tuberculously infected organism at a period when very little, or perhaps no, anatomical changes have yet taken place; by physical methods we are not yet in a position to demonstrate organic lesions and the clinical picture may still be quite negative.

expectoration, but in the rapidly progressive form, it is usually very profuse. It may also be copious in the chronic form if accompanied by much bronchiectasis, in which case with a sudden change of position the patient expectorates a large quantity of the sputum.

3. **Pain.** A variable quantity may be very slight or absent in some and very intense and pronounced in others. The pain is usually referred to as shoulder pain, as interscapular or as interscapular backache, pain in the second and third interspaces anteriorly, to a stitch in the side or a pleurisy pain, an apical or dry pleurisy, to myalgia or muscle pain in the pectorals, rhomboids, deltoids and intercostals and joint pain or arthritis as described by Poncet and pressure pain from contraction of the chest wall in chronic cases.

4. **Gastric Distress.** More or less pain in the stomach after eating. Except for the slight nausea which is usually present at the beginning of the disease, it is generally a late symptom. Vomiting may follow the ingestion of food. In the beginning there may be more or less constipation, which is often followed later in the disease by a toxic diarrhoea, a hypo-acidity being usually present.

5. **Dyspnoea.** This is a variable symptom. It may be slight or absent at first, or may be present on the slightest exertion. This may increase as the disease progresses and this is due usually to the lessening of the healthy lung surface. The respiration increases from 16 to 20 to 24 to 28 or more. Vagus irritation is a leading cause of dyspnoea.

6. **Hemorrhage.** This is usually a late symptom. (80) It may, however, be the initial one. A small mouthful of blood constitutes a pulmonary hemorrhage. A little blood or a blood streaked sputum is not a hemorrhage. We must be certain that the blood does not come from the nose, the mouth, or pharynx, teeth, oesophageal varicies, from heart lesions or from the stomach. A pulmonary hemorrhage is nearly always a sign of a cavity. A hemorrhage may follow exertion, a violent coughing spell or great excitement. In the old chronic cases there is a lessened tendency towards hemorrhages, in fact, slight hemorrhages in chronic cases is a more or less favorable sign, a sign of fibrosis and pulmonary contraction. A hemorrhage may be venous or arterial depending upon whether the blood is light or dark in color.

7. **Night Sweats.** Early or late. These are pathognomonic early symptoms as a result of fever and relaxation and are encountered in rapidly progressive cases, similating a crisis in the acute diseases. The CO_2 accumulation in the circulation may cause an irritation of the central nervous system and this is then followed by sweating. It may also be due to cardiac weakness, to the accumulation and reabsorption of secretions in the dilated bronchial tubes and to the lowering of the pulse during the night. Mechanical heat stasis due to too much bedding and clothing also may be the cause of sweating. The most favorable hours are between 3 and 5 in the morning, due to relaxation. All grades of sweating may be observed from slight perspiration of the face or hands to that of the half or even the whole of the body. The nervous apparatus, the cerebro-spinal anad sympathetic play a great part.

8. **Temperature.** This is the most important single (81) characteristic symptom. Fever is variable in type, depending upon whether the tuberculous process is acute or chronic, rapid or slow. Change in fever rate may be increased by exertion, working, walking, etc., by both physical and mental efforts, and may be decreased by rest and quiet. Fever is usually present in nearly all advanced cases, but usually not in the fibroid. Hectic fever, so-called, is due to mixed infection and absorption of toxins from pus and other organisms. Extension of the tuberculous process is always accompanied by an increase in the fever record. There is no arrest of the tuberculous process as long as there is fever. Fever is always an unfavorable sign. A temperature record of 104° is a very unfavorable sign, and one below 100 is favorable in tuberculosis. A sudden increase in temperature in the tuberculous is usually due to toxins of the bacteria of mixed infection, and less to the tubercle toxins. Absorption of the toxins is followed by lowering of body resistence. A rise in temperature may be preceded by a chill, or a chill and then a rise in temperature and then a drop. Usually the temperature is highest in the middle of the afternoon, normal towards the noon and evening hours and subnormal in the morning and early morning hours. In some inverse types, the temperature is highest in the morning and gets lower as the day goes on; again others have a steady slow rise reaching the highest point in the evening. These latter are more favorable cases, denoting fibrosis. The temperature here is usually within 100°. Again in terminal

cases the temperature may drop to normal and remain at that point from 6 to 10 days and even longer before exitus, or it may be alternately high and normal to subnormal. The temperature curve may assume the remittent type. This variable temperature may not be known to the patient, and as dissolution is approaching for hours, the temperature may be subnormal and accompanied by a slow, weak and thready pulse.

9. **Pulse.** Usually rapid. Tachycardia, an increase in the heart's action, a cardiac palpitation. The heart is usually normal while the patient is at rest. The rapidity of the heart is increased by exertion, excitement, emotions, while working or walking and after meals. The pulse is usually small, soft, compressible, of low tension and occasionally irregular, a symptom which is often very annoying to the patient. A systolic murmur at the base is frequently heard in beginning cases (hemic). A close relationship exists between the pulse and the temperature; if the pulse is too high in relation to the temperature curve, a bad prognosis is indicated. By the pulse rate we are in a position approximately to estimate the amount of lung involvement. In a tuberculous individual, a fairly constant pulse at 90 points to one-fourth of the lung being affected; a pulse of 100, half; 110, three fourths and 120, four fourths.

10. **Mental Symptoms.** The tuberculous individual usually shows much indifference and is very little disturbed. This is most particularly true in advanced cases. He is usually optimistic to the last. At the beginning of the disease he may show much irritability. Some are very melancholic, but few are pessimistic. Owing to our method of treatment necessary to control the disease, many become extremely selfish and dependent, have little will power, become lazy, but always remain hopeful and buoyant, giving evidence of extreme intoxication.

11. **Loss in Weight,** strength and courage with accompanying signs of pallor and anemia. The initial symptom in many cases is the recognized steady loss in body weight. The patient's skin begins to look pale and anemic and a feeling of tiredness accompanies this loss; the skin feels flabby and dry, the muscles show atrophy, bony prominences begin to show, fat disappears, finger nails look cyanotic, lips pale, eyes sunken, etc., accompanied by a gradual loss of ambition and force.

12. **Menstruation.** Amenorrhea, a frequent accompanying symptom in the tuberculous. Many cases of dysmenorrhea are

of tuberculous origin. Menstruation may again be regular after the 35th year of life. Proper hygienic measures may restore the normal menstrual functions. Usually with the cessation of the menses the tuberculous process increases, particularly in young adults. Ovulation may continue after the cessation of menstruation. It is important to observe, first the effect of tuberculosis on the menstrual period, and second, the effect of menstruation on the tuberculous process. See Chapter 26—Tuberculosis and Pregnancy.

13. **Hoarseness.** Usually a late, occasionally an early symptom. A change in the voice in some tuberculous patients is early noticeable. The voice is less resonant, feeble, hoarse, the vocal cords congested from coughing. Early hoarseness is generally due to thickening of the epiglottis; it may be caused by paresis of the recurrent laryngeal nerve, may be due to pleuretic thickening or enlarged cervical glands. Pulmonary tuberculosis in advanced cases is frequently followed by extension of the process to the larynx, producing a tuberculous laryngitis giving all the symptoms of pronounced hoarseness, accompanied by more or less severe pain on swallowing. Tuberculous laryngitis is always secondary to a tuberculous process in the lungs. It must be remembered that the pulmonary tuberculous individual may have as an accompaniment a laryngitis which is not tuberculous but simply a laryngitis acuta.

14. **Blood Pressure.** A lowering of the blood pressure is an important objective sign in the tuberculous. A low blood pressure has usually a correspondingly high pulse rate. The blood pressure varies greatly in the tuberculous if taken in the three positions, lying, sitting and standing, and here also a corresponding pulse rate can be observed. It is usually observed in the active tuberculous individual independently of age. The systolic blood pressure is never much above 100 mm. However, it may vary between 90 and 120, with a pulse pressure about 25. In the tuberculous developing much fibroid tissue or bringing about an arrest of the tuberculous process, a steady rise in the blood pressure will be observed. If the tuberculous individual has an accompanying nephritis, this will necessarily increase the blood pressure, due to the arterial changes, and again, if in a tuberculous individual with an active process, an increase of blood pressure is observed which is not accompanied by an arrest or at least

an improvement of his condition, an approaching hemorrhage may be anticipated.

(15) Other signs that are frequently observed in the suspected individual and which may suggest tuberculous disease and which if present should demand our closest attention are: (a) Fistulae-in-Ano; (b) chronic discharges from the middle ear (Otitis Media); (c) chronic fistulous tracts about the neck, the result of suppurating glands; (d) discharging sinuses about the body (psoas abscess); (e) long standing ulcers about the extremities (tuberculous ulcers, etc.).

CHAPTER 12

CLINICAL FORMS OF PULMONARY TUBERCULOSIS. CLINICAL VARIETIES

Grouping and Classification of the Various Forms

There are many different clinical forms of pulmonary tuberculosis which are so prognostically different that it becomes necessary from a pathological and more particularly from a therapeutic viewpoint to draw a sharp line between all these different forms. In considering the problem of pulmonary tuberculosis from an anatomical standpoint, we must remember that the manifestation of the disease as we see it is only a part of the disorder; there is usually a general disturbance, more or less pronounced, of all the different organs of the economy.

In order to give the pulmonary tuberculously diseased individual all the chances necessary for the restoration of his physical well-being, to bring about, if possible, an arrest of his diseased process, it will be imperative that the attending physician be perfectly familiar with all these pathological processes going on and from the pulmonary findings learn whether the process is active or stationary, and if stationary, whether there is a tendency to extension or a tendency at healing, or whether it is arrested or just beginning; upon the knowledge thus acquired he must base his prognosis.

All pulmonary tuberculosis said to be typical has its seat in one of the apices, and pulmonary tuberculosis beginning in any other portion of the lungs than in one of the apices may be designated as atypical.

Ever since tuberculosis has been studied as a distinct disease entity it has been observed that in about 90% of all cases of pulmonary tuberculosis the disease has its origin in one of the apices. This, being the general rule, may be considered as typical. As only exceptionally in less than 10% the disease has its beginning in other than the apices, these forms may be properly termed atypical, and the groups of these various forms of the disease, both the typical and the atypical, have many symptoms

in common. All forms may be classified as belonging to one of four divisions, namely, the atypical, mainly into a miliary, and the typical either into the exudative, proliferative or cirrhotic, depending upon the course the pathological process in the lungs pursues, whether more or less rapid or more or less protracted.

Clinical Nomenclature

Typical Types of Tuberculosis

Early in the pathological study of pulmonary tuberculosis it was observed that all the typical forms of the disease, those in which the beginning disorder had its seat in one of the apices, could be placed into either one of three groups depending upon the course of the tuberculous process. A simple classification (105) based entirely upon the pathological processes going on in the lungs and upon the clinical findings is best expressed by the following grouping or classification:

1. An exudative form. A rapidly progressing caseating broncho-pneumonia with a strong tendency to lung destruction.

2. A proliferative form. Slow developing proliferative changes with little or no tissue destruction, and

3. A cirrhotic form. A contracted chest, connective tissue hardening, with a slight tendency to extension but a strong tendency at healing.

A. **The Exudative (Caseous) Form.** (Caseation—Destruction, more or less diffuse lesions.) In this we notice a rapidly progressing caseating broncho-pneumonic process which is either acute or subacute, and like all other infectious inflammation of the lungs, consists of an infiltrate composed of leucocytes, a coagulable exudate, epithelial cells derived from the lining of the bronchiols, alveolar ducts, alveoi, etc. This inflammatory infiltrate undergoes rapid caseation and by contiguity causes more or less destruction of lung tissue and subsequent cavity formation. The extension of the process into neighboring tissue may be rapid, the produced caseous material aspirated into healthy areas, new tuberculous foci developed and the surrounding tissue being insufficiently aerated, breathing hampered and lymph flow harrassed, all producing conditions favorable to the extension of the process and growth of the tubercle bacillus and spreading of the tuberculous process, now more or less rapidly from above downwards.

Various observers have from time to time applied different names to this form of tuberculosis, all founded upon the course which the disease pursues during life. As all present a similar and more or less progressive picture and as in all an inflammatory infiltrate occupying the air cells is found, which is more or less moist, representing an exudate, the name of the exudative form of tuberculosis is the more expressive. The disease has been variously designated as: *Acute caseating pneumonia, tuberculous caseating broncho-pneumonia, pneumonic phthisis, pneumonic tuberculosis, acute or subacute pneumonic phthisis, caseous pneumonia, chronic catarrhal pneumonia, catarrhal phthisis, caseous phthisis, epithelial pneumonia, phthisis florida, acute peribronchial tuberculosis, tuberculo-pneumonic phthisis, acute disseminated peribronchial tuberculosis, chronic cavernous pulmonary tuberculosis, broncho-pneumonic phthisis, scrofulous phthisis, progressive caseating phthisis, galloping consumption, etc.*

Characteristic Signs and Symptoms. In this variety most of the physical signs are distinct and definite and the clinical picture that of a progressive, either slow or rapid, more often rapid and destructive pulmonary process. The fever is usually high, running a distinct curve high in the afternoon and a lowering in the early morning hours. Pulse is usually rapid and of low tension. There are many moist rales, a strong tendency to cavity formation: hemoptysis is frequently present, the sputum is usually bacilli positive and the stereo-roentgenogram shows more or less scattered coarse shadows and areas of distinct motteling. The exudative form of pulmonary tuberculosis especially at the onset (or a reactivating of a quiescent focus) is very frequently confused with pneumonia, both the broncho-pneumonic and the lobar form. The following are the chief points of differentiation.

Differentiation. (A) **From Ordinary Acute Broncho-Pneumonia.** The catarrhal or broncho-pneumonic form of pulmonary tuberculosis, acute exudative tuberculosis, is often clinically identical with and may simulate the non-tuberculous, acute broncho-pneumonia. The following are the chief points of differentiation. In acute exudative tuberculosis, the temperature is usually high, with a rapid pulse of low tension and a normal leucocyte count, a preponderance of lymphocytes and usually a positive tuberculin reaction, while in the non-tuberculous broncho-pneumonia the tuberculin test is usually negative, with an increase of the polymorphonuclear leucocytes from 15 to 30,000.

(B) **From Lobar or Croupous Pneumonia.** The differentiation between acute caseating or exudative pulmonary tuberculosis and ordinary or lobar pneumonia becomes at times very difficult, as both may be accompanied by bloody, rusty or clear bloody sputa and even by hemorrhagic nephritis. The signs of exudative phthisis, are the following: 1. The disease attacks usually robust individuals. 2. A history of tuberculosis in the family can seldom be elicited. 3. The onset of the disease is slow and insidious and is not preceded by a chill. 4. The premonitory symptom of a pulmonary tuberculosis may be a hemorrhage. 5. The fever usually runs a distinctly different course—is remittent in type, very different from that of pneumonia and is not accompanied by a crisis. 6. Generally the signs of dyspnoea and of cyanosis are slight or absent. 7. A progressive anemia of the skin and mucous surfaces is perceptibly noticeable. 8. The patient shows signs of progressive weakness. 9. The liver is nearer the spleen, but seldom enlarged. 10. The sputum is often greenish yellow, but may be blood streaked or pure blood, or may simulate the rusty sputum of pneumonia, tubercle bacilli are readily demonstrable and later with destruction of lung tissue, also elastic fibres.

B. **The Proliferative (Nodular) Form.** (Fibro-Caseous.) Here we observe a slower and more benign form of pulmonary tuberculosis and the involvement is limited mainly to the bronchiols and the alveolar spaces. In this form we find the typical changes with tubercle formation, epitheleoid and giant cells mainly about the lumen of the alveolar ducts near the junction with the alveoli. In this form lung tissue is not destroyed and elastic fibres remain intact. Very seldom, less by extension of the tuberculous process and chiefly through the presence of secondary organisms or caseating processes are cavities produced. In contra-distinction to the exudative form the development of larger or smaller nodules takes place, and tuberculous neoplasms or granuloma are demonstrable in the diseased areas. This form also shows a tendency to develop from above downwards and spreads by means of the respiratory bronchiols into neighboring and thence into other surrounding tissue. In this form we notice evidence of tissue irritation and reaction, due mainly to the action of the tubercle bacilli as foreign bodies. This form of pulmonary tuberculosis has been variously designated as:

Tuberculosis, Consumption, Incipient tuberculosis, Incipient phthisis, Chronic phthisis, Tuberculous phthisis, Nodular phthisis, Nodular tuberculosis, Chronic pulmonary phthisis, Chronic peribronchial tuberculosis, Chronic ulcerative phthisis, Tuberculosis sicca, Tuberculosis humida, Fibro-caseous pulmonary tuberculosis, etc. All these terms are synonymous, however, more definitely and appropriately expressed by "The proliferative form."

Characteristic Signs and Symptoms. A common form of the disease, more or less chronic, is usually characterized as apical tuberculosis. Two sharply defined groups of this form of tuberculosis may be recognized, namely, a dry and a more or less moist variety. In the former, secondary painful pleurisies and occasional friction sounds are very common, with the physical signs of harsh breathing, expiratory sound prolongation and intensification, note high and dull; crepitating sounds are not numerous or may be entirely absent, if much atelectasis, weakened breath sounds all showing a strong tendency at healing. This, the dry variety and with a good prognosis, is often referred to as the abortive form of tuberculosis, while in the moist group, assuming a position bordering on the exudative form, with occasionally slight destruction of tissue and the formation of small cavities and caseation, due to secondary bacteria, we find all the symptoms and signs as in the former variety, but accompanied by moist sounds, crepitant rales, and bronchial breathing, indicating a slowly progressive process which is often referred to as catarrhal tuberculosis or bronchial catarrh with a good prognosis. The physical signs are most frequently found above and below the clavicles, the supraspinous fossae and between the scapulae. The prognosis in this form of tuberculosis is always good, better in the sicca than in the humida variety. The third clinical group of pulmonary tuberculosis is:

C. **The Cirrhotic (Fibroid) Form.** (Fibrosis—Healing.) Lesions more circumscribed or discrete. Now connective tissue changes predominate, encapsulating and surrounding in the main the specific inflammatory tuberculous area. In the exudative or caseating form only slight if any connective tissue development took place, because the very rapid destructive process did not allow of encapsulation; in the proliferative variety this was somewhat in evidence but in this the cirrhotic form it is pronounced and definite. The conditions present in this variety

all favor the healing of the tuberculous process. The tissue outside and adjacent to the diseased bronchioles, alveolar ducts and alveoli is collapsed, followed by connective tissue formation, and this gives the area an appearance of healing in the center and an extension of the process at the border. Owing to the sluggish lumph flow throughout this fibroid connective tissue formation, large quantities of black dust particles and coal inhaled with the inspired air are deposited, giving these areas a dark appearance, and occasionally the bronchial tubes lying within this contracted area undergo bronchiectatic changes. To this form of pulmonary tuberculosis we also find that various names have been applied, all more or less indicating the formation of scar tissue within the lung structure, a strong tendency at healing and contraction, all of which is best expressed in the cirrhotic form of pulmonary tuberculosis. The following synonyms are used. *Fibroid phthisis, Cirrhotic phthisis, Chronic interstitial pneumonia, Cirrhosis of the lungs, Corrigan's disease, Induration of the lungs, Chronic pneumonia, Fibroid tuberculosis, Bronchitic tuberculosis, Bronchitic phthisis, Emphysematous phthisis, etc.*

Characteristic Symptoms and Signs. A history of the disease lasting for more than two years points to chronicity. The most evident mark of distinctiveness of this form of tuberculosis is the proliferation of tuberculous processes with the formation of scar tissue, a recession of the destructive tendencies of the disease, with accompanying symptoms of tissue change and secondary lung contraction, a vicarious emphysema, simulating asthma, much dyspnoea, little or no fever and no cachexia. The noticeable characteristic signs may be summed up as follows: 1. retraction and lessening of the intercostal spaces; 2. drooping of the ribs; 3. small angle at the junction of the ribs with both sternum and spine; 4. lowering of the shoulder on the affected side; 5. diaphragm high, immobile, and not influenced by breathing, (Williams' Symptom); 6. fixation of the chest on affected side; 7. lessening or absence of chest excursion; 8. the larynx and trachea are dislocated and with the heart and the great vessels, the mediastinum and diaphragm are drawn towards the contracted lung; 9. a high tympanitic, Wintrich's percussion note over the affected side localized near the sternum; 10. a definite roentgen shadow.

Owing to the lessened volume of the lung and decrease of air capacity, the percussion note is short and may have a tympanitic quality, but the vocal fremitus is distinctly impaired, and breath sounds usually harsh. If the lung is contracted over cavities or bronchiectatic areas bronchial breathing may be hard. In pronounced contractions of the lung, even of one lobe, there may be a heightened pressure in the pulmonary artery and consequent hypertrophy of the right heart.

The main object in practice is to separate these three different forms of pulmonary tuberculosis, to secure a clear picture of the condition of the lungs, so as to formulate a judgment as to a possible prognosis. Very little difficulty is encountered in recognizing the cirrhotic form. The duration of the disease for two years or more, retraction and smallness of the involved area, a sunken apex, a lowering of the shoulder, retraction of the intercostal spaces on breathing due to pleural adhesions, ribs prominent and drooping, diaphragm high, immobile, heart displaced, etc. Attention should here again be called to the percussion note which is usually high, due to a lessened volume of the lung and a consequent lessening of the amount of contained air with a more or less tympanitic quality like that over an infiltrated lung, but with a perceptible lessening of the vocal fremitus. As a rule on auscultation over a contracted lung, vesicular breathing of variable intensity is heard, and only in complete retraction over bronchiectatic areas or cavities is bronchial breathing audible. Difficulty in breathing due to pulmonary contraction is often followed by increased pressure in the pulmonary artery and consequent hypertrophy of the right heart, frequently developing a right heart insufficiency; such cases are surprisingly benefitted if treatment is directed towards the heart.

It is not difficult to differentiate the progressive, caseating, the exudative, as well as the proliferative from the evident cirrhotic form by simple physical examination, but much difficulty is often encountered in separating the exudative from the proliferative form, which are so prognostically different. In both, the percussion note is high with more or less tympany, or as in the beginning of infiltration only tympany similar to that of the cirrhotic form with a lessened lung volume and lessened amount of air is present, but on auscultation by complete infiltration bronchial breathing is heard, and if much of the lung tissue is still normal and air cells still free, the vesicular murmur is soft with

a slightly blowing expiratory sound. Moist rales possessing a specially high note are characteristic of the progressive or exudative form and the vocal fremitus is usually increased. The combination of the cirrhotic with the progressive infiltrative form with cavernous changes is difficult to separate. If over a contracted chest, indicative of fibrosis, one hears bronchial breathing, high crackling moist rales, the vesicular expiratory sound harsh and an increased or at least not a lessening of the vocal fremitus, then we can assume that upon the preexisting cirrhotic lung a progressive form has been implanted or that exudative changes exist in a cirrhotic pulmonary process. Another difficulty will be encountered in trying to separate the prognostically favorable proliferative form from the rapidly progressive florid variety. Here auscultatory and percussion differences are not distinct; however, a rapid progress of the disease bespeaks the latter form. If a whole lobe is involved and the duration of the disease is short, this would point to the exudative form, but a slow form of the disease may not exclude a rapid course because a proliferative process may take on a sudden activity and develop into the exudative. Again, the involvement of a whole or the greater part of a lobe speaks strongly for the florid form.

Further, we may observe the greatest variation in isolated cases in which the anatomical changes are insufficient to explain the process. In mild, benign, not very active cases one may find great toxic symptoms, high fever, rapid pulse, rapid heart, diarrhoea, emaciation and then requently in cases of very extensive involvement, fever may be entirely absent, appetite good with good nutrition and an absence of all general symptoms. The general conditions may be entirely different in two individuals and yet the anatomical process nearly alike. The number of tubercle bacilli demonstrable is also of no prognostic importance.

Atypical or Aberrant Types

In the above mentioned clinical classification those conditions alone were considered in which the disease had its beginning in one of the apices and which are generally known as the typical forms, namely, (1) and exudative form, (2) a proliferative form, (3) a cirrhotic form, leaving for our consideration the remaining or atypical forms which are only occasionally encountered and which comprise less than 10% of all varieties. These varieties

are usually comprehended separately as (4) (a) miliary, and (b) other atypical forms.

(A) Miliary Tuberculosis, Miliaris disseminata. Miliary tuberculosis is generally the result of a sudden inflow of tubercle bacilli in great numbers into the circulation. From the pathologic-anatomical character of the tuberculous lesion as observed in the organism, we may assume that very frequently and most readily tubercle bacilli may gain entrance into the circulation, and circulate in the blood stream. If, however, the entry is in small amounts, as so often happens, then the body is fully prepared to resist both deposition and growth. This is due to the existing defense agencies or antibodies within the body which were inaugurated at the first or primary infection. On the other hand, if the tubercle bacilli enter the circulation in large numbers, a massive involvement, or if in small but very virulent and often repeated numbers, or, as may happen, there is a sudden outpouring of the contents of a softened gland into the blood stream, then all of this may speak for a resultant miliary form of the disease. It should be mentioned here that the demonstration of the tubercle bacillus in the circulating blood not only in the miliary form of the disease but also in active and in advanced pulmonary tuberculosis is always a prognostically grave symptom—a signum mali ominis.

Miliary tuberculosis as we usually see it is a secondary process, the result of an active tuberculous focus somewhere within the body, an endogenous infection or more properly an acute auto-infection from a previously existing focus. Miliary tuberculosis as a primary affection is quite conceivable although improbable —must always be very exceptional, when we consider that the bacilli would have to enter the body in large numbers and from without. As to the method of miliary infection, we find that two views are held, namely, first that the bacilli find ready entrance into the various organs, but notably the lungs through the blood current, an hematogenous infection, usually in large numbers and overwhelming the system, producing as a result of this sudden inflow innumerable tuberculous nodules first in the lung structure because this is most vulnerable, this is soon followed by the invasion of the other organs; the second view is that the bacilli find their way into the blood stream in small amounts at a time, but often repeated, that these oft repeated introductions bring about the development of tuberculous nodules in the intima

of blood vessels, notably that of the pulmonary veins, and that subsequently these tuberculous nodules in the veins undergo changes, becoming soft and constantly pouring their contents in small amounts into the blood stream. This is very reasonable to assume, because in 95% of all cases of miliary tuberculosis these tuberculous nodules situated in the vessel walls can be demonstrated. This is also the more probable because these nodules are not found in the vessel walls in tuberculosis of the non-miliary form.

All forms of chronic tuberculosis, quiescent, arrested or active, may be followed by the acute miliary form if from any cause bacilli are mobilized and suddenly enter the blood stream in large numbers, hence all factors which favor the mobilization of the tubercle bacillus may bring about a miliary form of the disease. In youth and in young life such conditions frequently exist in an inflammatory infiltrate about the enlarged glands, in scarlet fever, in measles, etc., and in adult life we see it frequently following injuries, trauma or shock, in these latter without previous existing tuberculous disease in the vessel walls, both arterial or venous. We often notice that after injuries to enlarged and swollen glands, intestinal, mediastinal, cervical, etc., which rupture, their contents being poured into the circulation, after injuries or bruising of the testicle, epididymis, ovary or uterus, also after abortions, miscarriages, even after labor, a rapidly progressing miliary form of the disease has been observed. All this only goes to show, as I have so often emphasized, that throughout life, that is from the first infection in early infancy to the fully developed disease in adult life, throughout the whole process of the tuberculous disease, it remains a quantity disorder; it begins as a quantity infection and ends as a quantity disease, that is how many bacilli have entered either the body or the general circulation in a given time.

Diagnosis. Acute miliary tuberculosis has never been cured. We have no positive evidence that a case of acute miliary disease was ever followed by an arrest of the pathologic process, and all statements to the contrary must indicate a doubtful diagnosis. It is true that occasionally cases are met which assume a more or less chronic form, exacerbations and remissions following in rapid succession. The process is somewhat drawn out, but death is the inevitable result. Often the diagnosis from the objective symptoms presents much difficulty, especially in

children and the aged and here the pulmonary form may be suspected rather than diagnosed, and it may become necessary to resort to the use of the roentgen ray to confirm the findings. A preexisting tuberculous process either in an apex, near the hilum or in any other portion of the body may point strongly to a miliary form and examination of the blood for the presence of the tubercle bacillus may be necessary to clinch the diagnosis, that is, providing the examination is made early after the onset of the disorder, because the bacilli cannot be demonstrated in the blood if some time has elapsed since their entry. The diagnostic examination of the sputum will usually be bacilli negative, and this is to be expected as long as the tubercles are in process of formation. If, however, the disease has lasted for some time, as often happens in individuals who exhibit a slightly better resistance, then caseation and softening will take place, which generally occurs towards the close of the scene. Then if there is cough and expectoration, tubercle bacilli may be demonstrable. The disease usually terminates fatally in from two weeks to two or perhaps three months. The physical signs in miliary tuberculosis are, as a rule, not well defined but are more or less obscure. The percussion note is usually normal or hyperresonant. This is due to the distended air vesicles of the still normal lung structure and but for an old tuberculous process, healed or arrested, at one of the apices, the fremitus over the miliary involved area is not increased. Ausculation usually finds the respiratory murmurs harsh, with slight prolongation of the expiratory phase and râles, fine or crepitating, may or may not be audible. The physical signs being so much at variance with the clinical picture, this in itself should lead one to suspect miliary tuberculosis, particularly in an individual who for years has been sufferng from pulmonary tuberculosis which at times has assumed a more or less active form, or in a person who has tuberculosis of other organs than the lungs, like disease of the glands, bones and joints, of the testicles, ovary, epididymis, etc.

Symptoms. Miliary tuberculosis may have a sudden onset, very stormy, or may be somewhat protracted. There is usually headache, malaise, high fever, loss of appetite, great depression and prostration, and occasionally the initial symptom may be a hemorrhage. Then there is generally a dry, hacking unproductive cough, evidence of a bronchial catarrh with little or no expectoration, perhaps only a little clear mucous which at times

may be blood streaked. A most distinct symptom manifested from the very beginning of the disorder is shortness of breath, becoming gradually more and more distressing as the process progresses, respiration also more rapidly increasing to 40 and more per minute and with the progressing and growing infiltration of the lungs, cyanosis increases in severity. On inspection the lips and nails are found markedly cyanotic, the sclera slightly bluish and even the cheeks, which are found usually flushed, are slightly cyanotic. Fever is always high, ranging from 101-103 and even higher, the pulse rapid and feeble, the spleen is enlarged and tender and the urine is as a rule not free from albumin. Towards the close of the scene the patient may often manifest symptoms simulating a typhoid state with emaciation, delirium, dry and brown tongue, subsultus tendinum, etc. In miliary tuberculosis the symptoms often vary greatly, and may take on at any time the form of a typhoid, a pulmonary or meningeal variety, each of which has more or less definite signs and symptoms. In the suspected meningeal form, an examination of the chorioid becomes necessary, because in more than 75% of all cases from 6 to 10 tubercles, clear grayish yellow, round or oblong spots, can be recognized.

(B) OTHER ATYPICAL FORMS OF TUBERCULOSIS (44)

A very small percentage of all the various forms of pulmonary tuberculosis belong to this group. Some are generally highly toxic varieties of the disease. In the order of frequency are:

1. Typhotuberculosis (Typhobacillose of Landouzy) Tuberculosis resembling typhoid. Characteristic signs are an acute process, high fever, duration of the disease about four weeks attacking young individuals who are usually in good health. The fever is continuous, somewhat irregular with a corresponding frequency of the pulse. Examination of the blood indicates leucopenia and a relative lymphocytosis. Examination of the lungs at the onset is usually negative. Spleen enlarged. Evidence of involvement of the lungs, pleurae and mediastinum can later be demonstrated. Landouzy has pointed out that a patient suffering from this form of tuberculosis may frequently temporarily recover and that after a lapse of perhaps a month or more, a sudden and quick onset, a return of the disorder with great severity is noticeable, which is followed by speedy death.

2. Septic Tuberculosis. (Septicotuberculose of Neumann.) A

severe septic condition especially observed in cases of exudative phthisis, with great enlargement of both liver and spleen, jaundice, an inflammation of the joints, a generalized arthritis and tubercle bacilli are demonstrable in the circulating fluid. Both of these forms indicate a high toxic condition of the individual.

3. Tuberculosis accompanying Diabetes usually runs a very rapid course due to the favorable influence of the glycemia on the bacillary growth.

4. Tuberculosis following trauma may assume a more or less uncertain course.

5. Tuberculosis in the aged and in young infants assumes a more or less malignant and irregular course.

6. Tuberculosis secondary to the other forms of pulmonary disease, may assume a more or less atypical course.

7. Patients with either congenital or acquired chest deformities, like Scoleosis, Kyphosis, Kyphoscoleosis, etc., and who are very infrequently disposed to pulmonary tuberculosis, should they develop tuberculous diseases the lesions as a rule are not found in the apices but in the bases of the lower lobes or in the middle right.

THE DIAGNOSIS OF PULMONARY TUBERCULOSIS

Introduction into the methods of physical examination of the chest. Much depends in the treatment of pulmonary tuberculosis upon an early diagnosis. The earlier the disease is recognized in a given individual the better the prognosis. If pulmonary tuberculosis is a curable disease, and we maintain that it is, then the most propitious time for bringing about an arrest and probable cure of the tuberculous process is at the very beginning of the disorder and at a time when but little invasion or destruction of the lung tissue has taken place, and this usually is when the disease is still in the first or perhaps in the preactive stage, the stage of latency when the symptoms are still somewhat obscure; it is at this stage that we can promise our patients the most hopeful results. When once the disease is fully established, the prognosis can not be so favorable, although even at this stage with proper care and obedience, much good is often achieved.(41) (42) (46)

In advanced cases when the signs and symptoms are pronounced and the physical examination positive, when the layman

makes the diagnosis, an exact physical diagnosis and its interpretation are of interest only to the examining physician and to medical students for demonstrating the tuberculous process going on within the chest cavity, but they are of no value, use or help to the unfortunate tuberculous.

Every case presented for examination, irrespective of the stage of the disease, should receive full consideration; however, a beginning case should be studied with the most possible care and attention. First of all we must be able by physical examination to state definitely whether the individual is tuberculous or not tuberculous, for very much depends upon this.

If in doubt about your first examination, make a second, a third and perhaps a fourth at short intervals. There must be no doubt and we must state positively and with emphasis that the patient is tuberculous or that he is non-tuberculous. If by physical examination you have clearly demonstrated that the patient is positively tuberculous, then it is your plain duty to so inform him and tell him the truth; if, on the other hand, from your examination you have concluded that there is no tuberculous disease present, then again it is your duty to so inform the patient; this will relieve the mind and allay all fears and anxiety.

As previousy stated, the earlier the diagnosis is made, the better for the patient. In the examination of a patient, the methods of physical diagnosis by inspection, palpation, percussion auscultation and radiography must all be most carefully considered. In obscure or doubtful cases, to aid me in arriving at a definite diagnosis I have for years followed a plan which is well known to most of my students. All cases of doubtful or perhaps negative findings, after making a careful physical examination, are put upon a small iodide of potassium impression. I give the following prescription:

Iodide of Potassium, drachms two........................(4.0)
Tincture Cardamom compound, drachms four.........(8.0)
Peppermint Water, sufficient to make three ounces.........(90.0)

Mix. Direct that the patient take a teaspoonful in a mouthful of water once in three hours continuing this for three days.

The patient returns the third day and if on re-examination I now find constant râles over areas over which I found none at the previous examination, I carefully note this and make a diagnosis of probable pulmonary tuberculosis. An individual in whom such findings were demonstrable should return for a third

examination in about 8 or 10 days, and during this time he is to note carefully the temperature and pulse rate at least four times each day, 7 and 11 a. m., 4 p. m., and at bed time. If, however, after the iodide of potassium administration, the signs aré still negative, then I give intradermally a milligram of O. T. If this is followed by a reaction, I make a daily examination for three days, especially at the time when the reaction is at its height, and here also if over an area over which at a previous examination I found no râles I now find permanent crackling sounds, I make a diagnosis of presumable tuberculosis, but if after the administration of a Mantoux, there is no reaction, then I give a second intracutaneous injection of two milligrams. If after about four days this should also prove negative, then a third dose of tuberculin is given and no more if this also proved negative. I then inform the patient that in all probability he is non-tuberculous, and after this the radiographic chest plate may be helpful. Roentgenographic examinations should not be used primarily for diagnosis; they are only intended to confirm physical examinations. to check up the physician's ability to make the diagnosis by the physical methods.

HISTORY TAKING IN TUBERCULOSIS

Before entering upon the study of a case of suspected tuberculosis; that is, before we subject the patient to a thorough physical examination, it is above all necessary that we take a fairly accurate history of the case. A good history is to the clinician what a roentgenographic chest plate is to the roentgenologist, that is, a picture, a record of the case, and the more thoroughly this history is taken, the more clearly this mental picture will appear. Not all case histories are well taken, many are utterly worthless. Good history taking is quite an art and, like all things in practical medicine, it can be acquired only by persistent and constant practice.

History taking in Pulmonary Tuberculosis becomes quite a complicated affair,—there are so many angles to be considered. In looking over a case history sheet selected from any well conducted tuberculosis sanatorium, a striking feature is the long array of questions asked. This, in institutional work is found most satisfactory, although considerable time is usually consumed in tabulating the answers to these many questions. In general practice a history sheet must necessarily be somewhat

abbreviated, owing to the limited time allowed in each case, and yet a fairly good history of the case should be secured. Here then the outline must be more brief and to the point, yet in general, covering the case. This can fairly well be done according to the history outline as is given here: First ascertain the surname and the given name or names of the patient, city and street address, age of the patient, if single or married, vocation, occupation, weight, height, etc. Next:

Family History: Age of the father and mother if alive, if either or if both are dead, learn age at the time and the cause of death. Ask how many children there were in the family, whether all are alive and, if not, ascertain the cause and the age at the time of death. If there were many children in the family, ascertain what number in the group the patient is or, if a twin, about the health of the other twin; if dead, the cause and the age at the time of death. Inquire next about the father, if alive, if healthy, if dead. Is or was he subject to so-called winter cough? Or asthmatic, bronchitic, diabetic, etc.? Ask the same about the mother, perhaps either is or was tuberculous and the patient is quite familiar with this fact. Most particularly ascertain if there was in the household some one suffering from lung disease while the patient was in his early childhood days, when 2, 3, 4, 5, or 6 years of age. Quite frequently the disease is not acquired from the parents but from some one else about the household, and an old grandfather or grandmother, a male or female servant, a relative or friend living with the family is often the original source of infection. Make inquiry if during the tender years of the patient some one about the household was troubled with asthma, emphyema, chronic bronchitis, chronic cough, etc. Perhaps in these early years the patient for a shorter or longer time lived with friends away from home either in the city or in the country and a member of this household was suffering from pulmonary disorder; perhaps tuberculosis, and inquire if during his stay there he was very closely associated with this individual. As active tuberculosis is most frequently the aftermath of a close contact infection, particularly the contact of a tuberculous adult with the small child, the physician should be most specific to learn, how, when and in what manner this supposed infection took place.

Personal History: How long has the patient been complaining? What was the first thing that he noticed about his com-

plaint? If he had pleurisy, how recent or how many years ago? Ascertain whether he had measles, scarlet fever, whooping cough, pneumonia, typhoid, so-called walking typhoid or rheumatism. If so, when? Did he sustain any injury to body or mind, a trauma, automobile, railroad or any other accident? Any kind of shock? If an operation was performed, give the nature of the operation, was it recently or many years ago. Ascertain if alcoholic, if syphilitic or if gonnorrhoeal, if living quarters are hygienic, etc.

Present History, Present Complaint: What was the incidence that caused the patient to seek medical advice? The patient in all probability for some time, perhaps for months, has been complaining of a troublesome cough, possibly he had an initial hemorrhage, this has alarmed him and he now consults his physician. In other instances more obscure symptoms are the cause of his seeking aid, like headache, cardiac palpitation, nervousness, sleeplessness, gastric disturbances, extreme nervous irritability, malaise, shortness of breath, pain in the back or shoulders, etc., these almost always are the prodromal symptoms, or if the disease has progressed and activity is suspected, then more definite symptoms are present, for which he seeks relief, such as a warm feeling especially in the afternoon, acceleration of the heart's action, loss of appetite, perhaps night sweats, much coughing and expectoration, emaciation and loss in weight, etc.

Having secured a fairly good history of the case and before we proceed to a physical examination, it is in order that we note the pulse frequency, the number of beats per minute, take the temperature either by mouth or axilla or perhaps more accurate reading by rectum; note the number of respirations per minute and possibly take the blood pressure.

With these preliminary and necessary data tabulated we will proceed somewhat in detail to consider the various methods of examining the chest by inspection, palpation, percussion, auscultation and radiography, beginning with inspection preceded by a short note on and directions, "How to Examine the Chest."

CHAPTER 13

INSPECTION

How to Examine the Chest. Position of Patient. (34) (40) (43). The person to be examined should first be stripped to the waist, then comfortably seated, without any restraint, on a chair, or preferably on a stool, sitting erect. The chest must not be pushed unduly forward, nor excessively drawn in, the body in a natural, easy and comfortable position with the hands resting in the lap or the arms hanging leisurely by the side, or as some prefer the arms folded over the chest, keeping the knees close together, head erect and looking straight forward.

The examining physician is seated in an easy and natural, in no way cramped position, before and on a level with the patient, with knees pointing in opposite direction. In fact he must be seated perfectly comfortable. If he desires to examine the back, then either the patient must be asked to turn about or the physician takes his place behind the patient.

All necessary instruments like fever thermometer, percussion hammer, stethoscope, blood pressure apparatus, as well as writing paper, pen and ink, tape measure, tongue depressor, etc., should at all times be within easy reach.

If it be desired to examine the patient in the recumbent position, as is often the case in listening over the heart for faint lesions, then the patient should be placed in a comfortable position on the back, preferably on an examination table or chair. A bed may be used, but this is usually found too low and besides it does not meet the requirements of the examiner so well as does the chair for such examinations.

With the patient in the recumbent position either on a table, chair or bed, the arms should be placed at the side of the body or with the hands or arms resting on the head. Now with the patient in a comfortable and easy, either sitting up or lying position, the examiner at a glance must secure a relative picture of the external chest, if the chest appears normal or if there are any abnormalities and if so where.

(A) THE NORMAL CHEST ON INSPECTION

Inspection. Observe the general contour of the chest, whether both sides are symmetrical. The right side may appear normally a trifle larger, much depending on whether the individual is right or left handed. Note if the excursion over both sides is free and equal; one shoulder may be a trifle lower than the other, usually the right, all within normal limits. At the same time note the rate of breathing.

Next note carefully the different landmarks, first those on the anterior chest wall, then those posterior as well as lateral. If the chest wall anteriorly has been found fairly symmetrical then the landmarks will practically conform to normal conditions. First the clavicles will neither be over prominent on either side, the fossae above not very deep, nor very conspicious and no depression below (Mohrenheims fossa) but possibly a slightly hollow or shallow appearance. Ludwig's Angle "Angulus Ludovici," the junction of the menubrium sterni with the corpora sterni must not protrude too prominently and the ribs must not overlap each other, but flare out in a graceful manner alike on both sides, with but slight if any depressions between. The Pectoralis Major muscles will be equally prominent and symmetrical on both sides, and the attachment to the lower border of the 5th rib will furnish on both sides a more or less noticeable depression, "Sibson's Groove."

The area of that part of the chest wall in the nipple line from the 4th rib down to the costal margin and on either side should be fairly full and not show much depression or retraction, absence of "Harrison's Groove or Sulcus," nor a flaring upward and outward of the lower ribs, nor should much of a depression, if any, be noticeable below and about the xyphoid or ensiform cartilage, denoting absence of "Scrobiculus Cordis" and no depression above the xyphoid the "Pectus Excavatum" or funnel breast nor a sharp, protruding prominence of the breast bone, "Pectus Carinatum" like the keel of a boat, these if present, are all more or less abnormalities. The lower costal margin should not show a conspicuous flaring out nor should the costal angle be very acute and the junction of the costal cartilages and ribs show undue prominence, as is so frequently found in rickets or rachitis. The cardiac impulse should be normally visible or at least palpable in the 5th interspace and within the nipple line. The nipple, itself, particularly in the male, should be seen in

the 4th interspace about 4 inches (10 cm) on either side of the median line. In the female the presence of the mammae modifies somewhat the general appearance, but also here the general symmetrical condition on both sides must be maintained. Note also in a general way if the chest appears abnormally long from the shoulder down to the lower costal margin or if the chest is unusually wide from side to side as compared with the length.

While making these necessary observations about the chest,

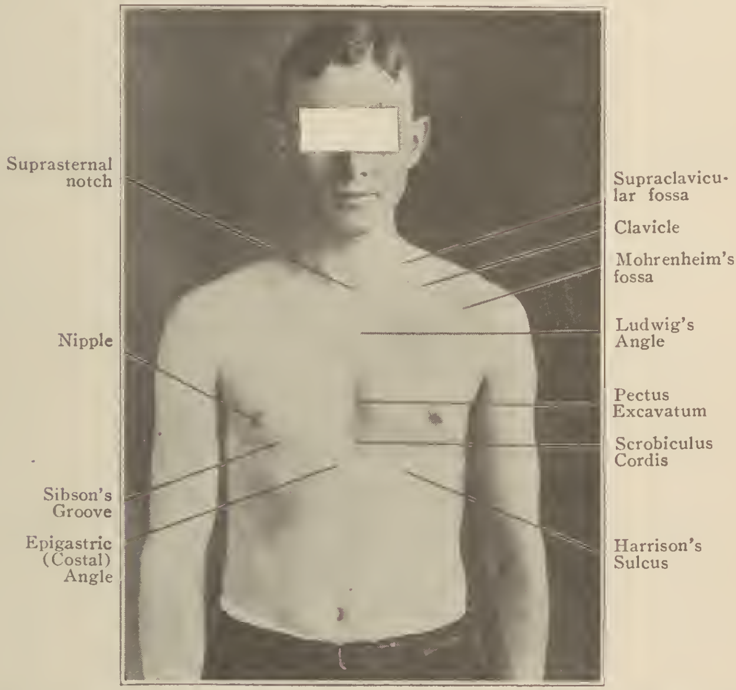


Fig. 9. The natural or normal landmarks of the anterior chest. These various landmarks if unduly prominent, either, protruding or showing a deep hollow, must be considered abnormal.

the examiner should incidentally note if the suprasternal notch is about normal or if abnormally deep or possibly entirely absent; if the thyroid gland is enlarged, if much or little, if some of the cervical glands are enlarged on one or both sides and about the jaw; if scar tissue evidence of former gland disease is present or not, if the arms are equally developed on both sides, if prominent veins about the chest, neck or arms are noticeable, if abnormal growths. Notice the condition of the face, and whether

the head is bald or has a healthy growth of hair, notice the color and appearance of the skin, of the eyes, if normal or otherwise. This whole picture must be reviewed at a glance and should not occupy more than a few seconds of time.

Having inspected the chest anteriorly, an examination of the posterior aspect should be made and the landmarks, mainly the scapulae and their relation to the vertebral column observed. Normally the scapulae are situated on either side and parallel to the spine and at about an equal distance from the vertebral

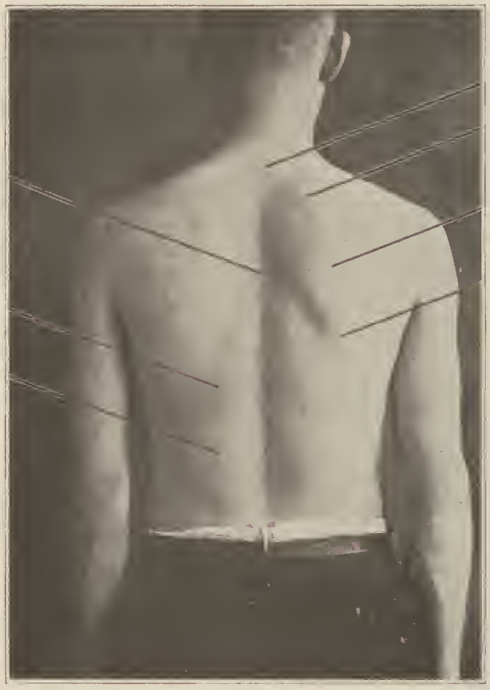


Fig. 10. The natural or normal landmarks of the posterior chest.

column. Has the vertebrarium its natural contour, neither deviating to the right nor to the left, absence of "Scoliosis," nor projecting unduly outward, absence of "Kyphosis" or being drawn noticeably inward, absence of "Lordosis," or perhaps one or more of the vertebrae may show a more or less sharp projecting angle of bone, presence of tuberculous disease, "Gibbus." Note the scapulae if they are an equal distance from the spine. Note the spine of the scapulae, if both are equal, if the depressions above the

spine are not unduly prominent and equal in appearance on both sides. Next note the lower angles of the scapulae, if they are equal or perhaps one more conspicuous than the other; then notice the area between the scapulae and along the spine, if symmetrical, if the muscular development of the rhomboideus the trapezius and the levator anguli scapulae, are alike on both sides and if the excursion of the chest is uniform and symmetrical over both sides. The appearance of the skin if healthy or if flabby, if veins or perhaps minute venules are conspicuously noticeable. Next notice if the lateral surfaces are symmetrical. A description or picture of the normal chest must not be taken too critically but only within certain limits, because we all know the difference in appearance of the chest of an exceedingly fat person as contrasted with that of a very thin individual. A chest to be considered normal must be fairly well symmetrical and that applies equally to the fat as well as the thin.

(B) THE ABNORMAL CHEST ON INSPECTION

Inspection. If in the ordinary examination of the chest much deviation from the normal has been observed, or if various parts show some abnormality, then a special and more careful examination is required. This brings us to a consideration of abnormal findings on an inspection of the chest, the picture as being presented at a glance, a first view, or what is generally described as Prima Vista Diagnosis.

General consideration. In basing a diagnosis on inspection it becomes necessary that all the various methods of examination be made available, for instance, the relation of body weight to height, the circumference of the chest, etc. In a large number of chest examinations made, and noting the average, it has been observed that not only does the individual suffering from active pulmonary tuberculosis and its complications show a perceptible loss in body weight, but that many persons who possess a subnormal body weight and are not already suffering from tuberculous disease become easy prey to its ravages. Attention should be given then first of all to the circumference of the chest in the adult. At birth and for years thereafter the general appearance of the chest is more or less cylindrical assuming towards puberty and remaining for years thereafter the oval type, to resume again in old age or reverting back to the cylindrical or "infantile" type. In the normal adult the chest is oval

in outline, the antero-posterior diameter is less than the lateral in proportions nearly as is 1 to 2. In the individual suffering from long standing or chronic pulmonary tuberculosis and in those predisposed to the disease, the chest is usually flat antero-posteriorly, wide laterally and long from above down. Again, the relation of body weight to height; in the average normal male for every foot in height 25 pounds should be added, and in the female about 23. A man whose height is 5 ft. 6 inches, (168 cm.) should weigh within 138 pounds (65 kilos). In the person suffering from tuberculous disease, either active or of long standing, this relationship is often greatly disturbed. Next note the circumference of the chest to body height. Normally the circumference of the chest in inches should be at least half that of the body height. A healthy individual whose height is 5 foot 6 inches (168 cm.) should have a chest excursion of more than 33 (84 cm.) inches or at least 33 (84 cm.) inches when at rest. These variations must always be great and can not be applied in all individual cases and are considered more or less crude methods in diagnosis; nevertheless they point to abnormal chest probabilities.

The chest on inspection may show definite signs of an existing disease. This is often noticeable, and particularly in children. It may present evidence that the disease has existed at some time and that the process is now healed, or that the formation of the chest is such that the individual who possesses such a chest falls easy prey to tuberculosis. It may also give evidence that the possessor of such a chest early in life came in contact with tuberculous disease but has weathered and braved the storm. hence from these observations we assume that certain signs in an individual may stigmatize the possessor as being classed as tuberculous, either past, present or future.

Stigmata of Tuberculosis. Early in the study of the subject of pulmonary tuberculosis, the careful and observing physicians were impressed by the facts that certain individuals or even groups of individuals, possessing chest formations which showed a more or less distinct type, were either already suffering from tuberculous disease or later developed a tuberculosis, and these types were described as having many signs in common and yet were capable of a distinctly separate classification.

(A) **Chest Deformities which point to a tuberculous disease, past, present or future—The chest as a whole. (44)**

1. **The Stenotic or Contracted Chest.** Children and adults as well possessing this type of chest are predisposed to tuberculosis. In this form of chest development we find the upper aperture, that is the first rib and its cartilage, small and contracted. The first ring is small, the cartilage also small and early ossified, and the rib is but imperfectly developed. This causes contraction of the apices and deformation, accompanied by early stasis, insufficient blood and lymph supply with faulty aeration. This type of chest offers a mechanical disposition to a tuberculous disease which is manifested by two clinical varieties, according as the cause is a disturbance in the development and contraction of the above described first rib and cartilage, or is a congenital or more or less acquired scoleosis of the upper spine in the paravertebral area. This type of chest formation which was first described by Freund and further enhanced by the labors of his pupil, Hart, is now recognized by most observers as a distinct form of the chest predisposing to tuberculous disease. Classic pictures of this form of chest development are frequently seen in art galleries, and paintings, portraits and figures of the fair sex, depicted with long, scrawny and conical necks, small upper chests are of this type.

That the contraction of the upper aperture incident to early ossification of the usually small cartilage of the first rib, and the faulty development of the rib itself are important contributing factors in tuberculosis is now generally recognized. In an intensive study of 394 roentgenographic chest plates, 221 of which were those of male and 173 of female patients, ossification of this first cartilage was demonstrable in 58%, and in addition in 48% it was possible to show definite tuberculous shadows in the lung structure, and it was also shown that the frequency of ossification of the first cartilage increased with age and that in youth only in about 10% was this present and then usually unilaterally, becoming more bilateral with mature years, all proving that tuberculous disease has a special predilection for the apices if the first rib and the ring are small and the cartilage is early ossified.

2. **The Thorax Infantilis, Infantile type of Chest.** In this form of the chest, predisposing to tuberculosis, the round form, common in infant life is still retained. In children and young adults possessing this type of chest, the gradual change from the infant to the adult type has not taken place, the infantile form still

maintaining, and possessors of this form of chest are very prone to pulmonary tuberculous disease. In this form, as in the preceding, faulty aeration plays a prominent role, and enlarged tonsils, adenoid vegetation, obstruction to the upper air passages, insufficient circulation and aeration, accompanied by rickets, drooping of the ribs, narrow intercostal spaces, large lower aperture and possibly a high palatal arch generally completes the picture. This type predisposes relatively less frequently to tuberculous disease than does the former.

3. **The Phthisical Chest—Thorax Phthisicus, seu Paralyticus, Habitus Phthisicus, Habitus paralyticus.**

The long, narrow and flat chest. A close relationship to heredity, a simple form of the hereditary chest, and the chest deformity which gives evidence of a previous tuberculous process, a tuberculosis having preceded or accompanied this type of chest development. This form of chest, however, gives no absolute proof of any connection between the phthisical chest and tuberculosis, because, as is well-known, tuberculosis is often found in individuals with perfectly normal chests; nevertheless an etiological connection between this form of chest and tuberculosis must be admitted from the fact that pulmonary tuberculosis develops surprisingly rapid in chests hindered from free and full expansion because here the function of the lungs is hampered by physical disturbances, and such an unexpanded and illy ventilated lung, particularly that portion in the contracted upper aperture offers a nidus for the growth and development of the tubercule bacillus. The classic picture of this form of the contracted chest is due to a deficient blood and lymph supply and insufficient aeration, thereby furnishing a very favorable mechanical disposition to a tuberculous infection, which is early followed by active tuberculous diseases and speedy death. Thorax phthisicus embraces more or less nearly all the stigmata of the stenotic and infantile type, with the addition of loss of fat, muscle degeneration, emaciation, cachexia, etc.

The chest deformity generally known as "Habitus Phthisicus" was very early recognized and is even at length described in the writings of the ancients, who at that early time had noticed the fact that this condition predisposed to early tuberculosis. Such individuals are usually thin, slender and scrawny, sitting and walking much stooped, have but little if any adipose tissue, muscles are poorly developed and flabby, with a dry and pale

skin, throughout which, mainly over the anterior and upper posterior chest, minute venules are noticeable. The neck is thin and long, cheeks hollow and flushed, the chest flat and long from above down, and narrow from side to side, hands and arms emaciated, the face presenting a tired expression, and with uually long lashes the eye presents that condition known as the appealing eye.

(B) General Observation. Suspicious signs pointing to tuberculous disease, past, present or future.

Prima Vista Diagnosis.

A first view impression or diagnosis. In itself a crude method, nevertheless pointing to some abnormality. The picture which impresses itself upon our mind when we see or examine the chest for the first time. If at this examination the chest is found abnormal, especially the chest of the youth, it may correspond to one or other of the three types described above, or it may be a composite of all. Most signs as observed are those of a chronic condition of the lungs, healed or arrested lesions, or those of a functional disturbance consequent to a faulty chest development. However, abnormalities are also observable in some instances at the beginning of the pulmonary tuberculous process, particularly that of the slow or benign form. Singly, these signs may be of no value, but in correlation with others they bear evidence that at some time in the life of the infected individual a pulmonary disturbance, functional or otherwise, has taken place. The following are some of the more important signs now generally recognized, a few of which have been previously mentioned in connection with the abnormal chest types described above.

1. Chest measurements:

a. Abnormal length. The measurement of the chest from the neck above to the costal margins below (the cranio-caudal measurement) is usually greater than normal (is longer).

b. Abnormal diameter. The measurement of the antero-posterior diameter of the chest (The sterno-spinal measurement) is generally less than normal (is shorter).

c. Abnormal circumference. The circumference of the chest as compared to body length is smaller than normal. (The circumference should be at least one-half the body length.)

2. Kyphosis. Slight or pronounced. Probably also a scolesosis (Kypho-Scoleosis) mainly of the upper dorsal area, may be present.

3. Conspicuous and prominent protruding Ludwig's angle. (Angulus Ludovici prominens.)

4. Drooping of the shoulder on the involved side.

5. Early ossification of the first cartilage and a secondary shortening of the first ring already described above.

6. The clavicle. Abnormalities of this important landmark are most interesting. (54) The clavicle as a whole may be much displaced, may be very prominent or project conspicuously, or may remain fixed on inspiration. This usually is due to shrinkage of the cupula pleura and adhesion to the first rib. In chronic apical tuberculosis of one lung only the clavicle on the healthy side may be perfectly normal, showing a distinctly contrasted appearance as compared with the diseased apex. The sternal end of the clavicle may be noticeably lower over the affected than on the healthy side. This can be readily demonstrated by placing the index fingers of both hands on the sternal end of the clavicles when the difference between the healthy and the diseased side can be recognized. On the affected side the clavicle is lower. The acromial end may be much more prominent, showing many bony prominences and deep depressions above and below, as compared with the opposite, if healthy side, and the body of the clavicle itself as compared with its mate on the opposite or healthy side may be thicker than normal.[1]

7. The supraclavicular and infraclavicular fossae. In old, chronic, healed or arrested cases, the fossa above the clavicle will be found abnormally deep as compared with the opposite side. If the other side has not been the seat of disease, the contrast will be particularly noticeable. Usually in such cases the depression below the clavicle (Mohrenheim's fossa) will also be more prominent.

8. Visible retraction, immobility, emaciation, muscular atrophy and flatness over the affected area.

9. The intercostal spaces on the affected side will be found deep and narrow and the ribs overlapping and drooping.

10. The cardiac impulse may be diffuse, rapid, circumscribed, or visible to the right of the sternum or displaced upwards.

[1]This thickness of the body of the clavicle and often of the sternal portion as well bears a close relationship to an old tuberculous process in the apex. If both apices have for some time been the seat of tuberculous disease and the involvement of one has been secondary upon a slow and chronic process in the other, then the clavicle over that apex which was the seat of primary lesions will be the thicker. Also the clavicle on the affected side will be found to slope more than its mate on the healthy or opposite side.

11. The epigastric or subcostal angle. This angle which in the normal chest is usually broad and wide, filled by the abdominal muscles and the viscera in the tuberculous chest is found to be narrow and high, showing more or less contraction of the soft parts, giving the costal margins and the ribs usually a prominent appearance.

12. The tenth, a floating rib. Costa fluctuans decima. The ribs from the first to the sixth are attached to the sternum each by its separate cartilage and the seventh, eighth, ninth and tenth by a common cartilage. In some individuals this tenth rib is not attached to this cartilage, is free from all connection with the sternum, similar to the eleventh and twelfth, hence such persons possess three floating ribs instead of only two as is the general rule. According to Stiller, people with such an abnormality generally suffer from tissue relaxation, nervous dyspepsia, neurasthenia, from enteroptosis, gastric immotility, the stomach is never empty and splashing sounds are often elicited, in short, they suffer from general asthenia, a constitutional anomaly, and consequently fall easy prey to accompanying tuberculous complications (a habitus asthenicus.)

13. The Scapulae. The scapula on the affected side may show undue prominence and flare outward, giving it a winged appearance, the scapula alata, or may approximate closely towards the median line. In the beginning of the tuberculous process the rhomboideus may be in a state of spasticity and this condition has a great tendency to push the scapula outwards; with the continuation of the disease muscle relaxation follows in the usual train, the muscle degenerates and having lost its tone the scapula now lies passively by the side of the spine. This again denotes an old or chronic process.

14. Respiratory Excursion. A more or less pronounced lagging of the affected side on deep inspiration is noticeable in all chronic forms of tuberculosis. In a recent tuberculous involvement, lagging of the affected side will be often observed if an inflamed pleura mechanically interferes with free breathing, the patient suppressing the respiratory movements on the affected side as much as possible.

15. Veins and Venules. Enlarged veins over the anterior chest wall, (compression of vena azygos) prominent venules posteriorly on the upper chest on one or both sides of the spine and above the spine of the scapula and over the anterior chest on

either or both sides of the median line over the 6th, 7th and 8th ribs and interspaces and towards the axillae are evidence of pulmonary stasis. If veins are enlarged or venules prominent, unilaterally they present strong diagnostic evidence of either pulmonary disease or mediastinal gland enlargement.

16. The Mammae and the Nipples. In the male the nipple is found in the 4th interspace normally, and with contraction of the chest, consequent to tuberculous disease, the nipple may be displaced. The mammae in the female modifies conditions, depending upon the size. Here, usually, we find the mamma on the affected side smaller and less developed than the one over the healthy side, the nipple itself is smaller and less fully developed and the areola also is smaller and is less pigmented over the affected side.

17. The Pupil. The pupil is often dilated on the affected side. Apical pleurisies accompanying apical tuberculosis, or enlarged tuberculous glands causing irritation or pressure upon the fibres of the sympathetic nerve, is usually the cause of this dilitation.

18. Red Line along the Gums. (Steger) As evidence of toxicity. A red line is sometimes seen in tuberculous individuals following the gums, similar to the blue line in lead or the black line in bismuth poisoning. Its significance is not well understood.

19. Muscular Spasticity and Atrophy. (Pottenger's Sign.) In beginning tuberculosis the various muscles of the upper chest, those overlying the pulmonary tuberculous process, like the trapezi, the pectorals, the sterno-cleido mastoids, the Scaleni, the rhomboidei and others are often found in a state of spasticity. This muscular rigidity persists until the tuberculous condition has lasted for some time, when a change in the muscle substance takes place and the fibers undergo degenerative changes which in chronic cases are evidenced by distinct atrophy clearly visible on inspection (and by palpation as well).

20. Finger Nails. Distinct trophic changes are often noticeable in most cases of chronic pulmonary as well as cardiac lesions. In pulmonary tuberculosis which is assuming slowly the chronic form, distinct changes are observed, which begin with a slight hyperemia and then thickening and glistening of the nail matrix. The nail then gradually assumes an antero-posterior curve similar to the lateral, a gradual thickening of the

bulbs of the finger ends until the fingers are curved, cyanotic, and enlarged, being then described as "clubbed" fingers.

21. Skin and Hair. The skin in the tuberculous person has also undergone some changes, taking part in the general trophic disturbance incident to the disease. The skin is usually pale, and anemic, or slightly yellow or even brown and lichens, pityriasis and psoriasis are not infrequent. The skin is dry to the palpating hand, has lost its moist and unctuous feel, is somewhat scaly, and may even feel feverish. The hair on the chest seems to appear disordered, irregular in growth, in children downy, showing a lack of luster, evidence of faulty nutrition and trophic changes.

22. Mouth. In examining the mouth by inspection, one is often impressed, in young individuals suffering from tuberculosis, that the palatal arch is unusually high. This is akin to those nose and mouth disturbances incident to enlarged tonsils, adenoid vegetations, or hypertrophic turbinates. These obstructions to the upper air passages often bring about faulty aeration, particularly to the apex of right lung and thus favoring tuberculous disease. In addition, in active pulmonary tuberculosis, the uvula, soft palate and pillars are often pale and anemic.

23. **Litten's Phenomenon.** Absence of the phrenic shadow, or Litten's Sign, a diminution in the excursion of the diaphragm on the affected side of the tuberculous. The phrenic wave, the peeling off of the diaphragm during inspiration. If a person with normal chest, lying on his back, chest bared to the waist, feet pointing towards a window, is asked to inhale, the examiner standing at the patient's side will observe a narrow shadow in the axillary line from the seventh to the tenth rib, about 2 to 2½ inches of excursion, moving down with inspiration and again up with expiration. The absence of this shadow on either or both sides indicates some previous disturbance in the pleura or lung. In beginning tuberculosis the shadow is often diminished or absent owing to the lessened elasticity in the affected lung and probably also to pleuritic thickening and adhesions.

24. **Integument Atrophy.** When an apex has become (64) tuberculously invaded, a peculiar phenomenon manifests itself as soon as the process is well established. Nature, here, as in every other form of tuberculosis, endeavors to put the parts at rest, and we notice that the fascia, subcutaneous fat, muscles, (Panniculus adiposus et carnosus), and integument, give way

early to trophic changes. If, over the anterior chest wall in the parasternal line, we pick up the overlying skin with the thumb and middle or index finger and pull it towards us, we very readily observe that the tissue has entirely lost its elasticity, the skin feels lifeless, and we quickly notice that the amount of skin held between thumb and finger is perceptibly less when compared with the other, perhaps still healthy side. If both sides are the seat of tuberculous involvement, the greater amount of atrophy

Fig. 11. Integument Atrophy. (Demonstrating Integument Atrophy.)

will be noticeable over the apex which was the seat of primary disease. (This is particularly noticeable when we combine Inspection with palpation.) Wheaton directed attention to this interesting phenomenon as early as 1910.

25. **The phthisical facies.** Last, the facial expression of the phthisical subject, the usually characteristic oval face, the pinched features, the dilated pupils, the whitish-blue sclera and the appealing eyes must be mentioned. In such a picture one can readily observe in one's mind the rapid, shallow, unconscious breathing all bespeaking the gravity of the disorder.

CHAPTER 14

PALPATION

Definition: The word Palpation as defined means to feel with the hands or with the tip of the fingers, the laying on of the hands; in practice it is the application of the examiner's hand, hands or fingers over various parts of the chest wall of the patient, the patient being asked to speak at the same time. It is next to inspection the most important method of physical examination when the hand is placed flat on the chest wall of the person to be examined and the patient is then asked to speak or count out loud, a peculiar vibratory sensation is communicated to the palpating hand. This is usually described as fremitus, thrill or tactile fremitus or more correctly as vocal fremitus. Normally, if the palpating hand is applied, this vibratory murmur is felt with more or less intensity over certain areas and entirely absent or nearly so over others. All things being equal, in palpation much depends upon the voice of the individual to be palpated. A high pitched voice will give little or no fremitus whereas a low pitched voice will produce a fremitus which is palpable over the greater part of the whole chest and it is because of the voice that women and children usually give less vocal fremitus than do men. The more sonorous the voice the greater and better the vocal fremitus and vice versa.

Methods of Palpation

The simplest method of practicing palpation, the one most in vogue, is by applying the palm of the hand firmly against the chest wall, at the same time asking the patient to count aloud and repeatedly, 1, 2, 3 or 99 or 66. It is essential that the hand be placed firmly against the chest wall, allowing no hollow space between the palpating hand and the thoracic wall. Another method in great favor with many is to palpate with the lateral margin of the hand along with the little finger while the patient counts or speaks. Still another method and one which I consider of particular importance because of its subtle manner is finger palpation. This is practiced, and is particularly applicable in the examinations of small infiltrated areas in the apices of the

lungs, by placing the thumb of the right hand over the right upper and posterior lobe of the lung of the patient to be examined, the thumb of the left over the left upper, the right and left index finger in the right and left supraclavicular fossae, respectively, and the middle finger of each hand placed in the depressions below the clavicles. If the patient now is asked to speak while the fingers are in this position, a peculiar but definite sensation is imparted through the palpating fingers. This is especially acutely noticeable over the upper and posterior chest through the palpating pulp or ball of the thumb. Another method of palpation often practiced is by grasping the upper apices posterior with the ball of either hand resting firmly against the muscular tissue above the spine of the scapulae, the fingers firmly grasping the tissue above or below the clavicles. In practicing palpation by this method, a method in high favor by many lung specialists, while the patient is asked to speak, the apices are firmly grasped, at the same time making slight pressure with the palpating fingers toward the ball of the hand. All the above mentioned methods are usually known as immediate palpation in contra-distinction to mediate palpation, that is by means of a media between the chest to be palpated and the palpating observer. Mediate palpation is also known as osteal palpation, and for sensitiveness as a method of palpation it has no equal. Mediate or osteal palpation is generally practiced by the examining physician, either sitting or standing behind the patient, placing the hand over the upper chest, over the apex, and then placing his forehead directly and firmly upon his hand, at the same time asking the patient to speak or count aloud. By this method a distinctly definite and sharp vibratory sensation is communicated to the forehead through the hand and even the slightest variation in the two sides can most readily be recognized by this method. Fremitus is often described as subjective and objective; subjective as observed by the patient himself and objective, that which is elicited by the palpating hand of the physician.

Palpation of the Normal Chest. As a coarse, sonorous voice will give an amplitude of sound waves of greater intensity and larger volume than a high and feeble voice, it becomes evident that the fremitus must be greatly influenced by different voices. The fremitus is also greatly influenced by the thickness of the muscular development, by the amount of adipose tissue, whether

the chest to be examined is that of a fat or fleshy or that of a thin and scrawny person, whether the chest be that of a child or of a woman or that of a man old or young, hence, the voice, the sex, the age, the chest formations, all must be considered while practicing palpation.

In placing the palpating hand on the normal chest, the vibratory impulses imparted to the hand differ over different areas. Usually the fremitus is most pronounced and definite over the right chest, both anterior and posterior, and over the right apex more than over the other portions of the lung, and over the left lung the fremitus is usually much in abeyance or may be entirely wanting or only slightly felt over the apex. Various reasons are given for the greater fremitus over the right as compared with the left side. (1) The greater number of right handed people and thicker muscular development; (2) the larger lumen of the right bronchus; (3) the more direct access of air and sounds to the right upper lobe; (4) the usually somewhat contracted upper right lobe, the right apex being more conical and the left more dome shaped (see Figs. 14 and 15); (5) the right lung lies in closer proximity to the trachea and larynx than does the left; (6) the large organ at the base of the lung, the liver, acting as a sounding board, etc.

Palpation of the Abnormal Chest (17). The normal lung, as we observe it, is usually a poor conductor of sounds, and anything which will replace the air contained in the alveolar cells, bronchials and small bronchial tubes by solid or semi-solid material will convert it into a better conductor. If the parenchyma of the lung becomes airless, the normally inhibitive influences of the sound conveying conditions are lost and in place of a poor sound conducting medium we now have one which is a fairly good conductor, the conduction depending more or less upon the amount and extent of airless lung structure. The more intensely airless this air contained area is made, the more pronounced is the tactile or vocal fremitus, and anything which will make a good conductor out of the normally poor conducting lung tissue will in that proportion increase the fremitus.

In palpating a lung or an apex to ascertain the extent of infiltration or involvement of the underlying lung structure, it is equally important that in addition to observing the fremitus we note also most carefully the amount of resistance. An infiltrated condition of the lung will generally be accompanied by a vocal

fremitus and an increased muscular resistance, and if an area of the thorax which under normal conditions is negative as to fremitus should give an increased resistance and a vocal fremitus, that is always a sign of a pathological lesion. The fremitus being normally greater over the right upper lobe, if in a given case the fremitus is greater over the left apex or if only equal to that of the right or apparently equal, then we have positive evidence of left sided apical involvement, and if, in addition, the palpated muscles are more rigid on the one side and more relaxed over the other, then we most likely have additional evidence that on the one side over the rigid muscle the process is probably active whilst on the side where the muscle is relaxed the process is old or arrested. If an apex is infiltrated only recently, is not a very old process but an active one, then all the muscles over and about this active area are in a state of spasticity, and we find the sterno-cleido-mastoids, the scaleni, the intercostals, in a less degree the pectorals anteriorly, and posteriorly the trapezi the rhomboidei, the levator anguli scapulae all more or less resistant to the touch. This spastic condition may last for some time, being more prolonged if the pulmonary process is slow, but ultimately the muscle rigidity will lessen and later degenerative changes will appear, and then to the palpating finger a sense of lessening of resistance, a lack of muscle tone, is imparted until gradually the muscle becomes more and more relaxed and now nothing remains but a few fibrous bands. The muscle spasm (35) or rigidity noticed in many conditions of acute inflammation of various organs of the body, for instance, the rectus over an acute appendicitis, the various abdominal muscles over an acute pancreatitis and the muscle spasm so frequently observed in acute bone and joint disease are all of like nature. In these conditions the muscle spasm is more readily observable because the onset is more acute, is sudden, whereas in a tuberculous infiltration of an apex the process is slow, protracted, even chronic and insiduous, and the condition of the overlying muscles often goes unnoticed.

In pulmonary tuberculosis which in the greater number of instances begins in the apices, the right more frequently than the left, an increase of the vocal fremitus is most important in diagnosis. If an individual with an apparently normal chest and pitch of voice not too high or low be asked to count 1, 2, 3 or repeat 33, 66 or 99, a very distinct fremitus is conveyed to the palpating hand or finger, and if while comparing the fremitus of

one side with that of the other we notice a distinct difference in the normal relationship of the two sides, if there is a plus fremitus, a fremitus more than normal, and if at the same time the overlying or contiguous muscles are in a state of spasticity or rigidity or perhaps the muscles over the side where the fremitus is increased have already become soft and relaxed, again we have evidence which points to a tuberculous process active, recent, old or arrested, depending upon the area over which we find spasticity or degenerative changes. Fremitus over the base of the lung is felt most distinctly when the lower lobe, either right or left, is the seat of inflammatory changes, and here also muscle resistance and relaxation are noticeable, and the intercostals being either stiff and spastic or distinctly relaxed and soft, depending on whether the pulmonary process is early, recent or late.

Painful and tender points. In some instances of beginning pulmonary tuberculosis as well as in some cases in which the process has existed for some time, perhaps a long time, especially if the seat of the disease is in one apex, pain can be often elicited by the palpating finger. Begin palpation with the tip of the index or middle finger at the lower angle of the scapula and by making gentle pressure follow along the inner margin, upwards to the spine of the scapula, and in the course a point may be reached which is more or less painful. If, while making light pressure, a strict watch is kept to see the expression on the face of the individual being examined, we can often observe that when a certain spot is reached there is a sudden expression of pain, the person seeming to flinch. The painful spot is usually permanent, is usually posterior over either the right or left apex or it may be demonstrated over both apices. With the palpating finger following up along the scapula, first on one side then on the other, this phenomenon can easily be elicited. This sensation of pain, hyperesthesia or algesia, the demonstration of which is described as algoscopy, is usually due to an apical pleuritis which often accompanies the tuberculous process.

In the early beginning of tuberculous infiltration, due to the collapsed condition of the air cells surrounding the initial tuberculous process, the vocal fremitus may be somewhat decreased as it is also in the old and chronic form of the disease when the pulmonary tuberculous process is accompanied by much contraction. The fremitus also is often found to be decreased owing

to the emphysematous condition of that portion of the lung surrounding the contracted tuberculous area.

If while palpating the chest of the patient, he has a fit of coughing, then frequently a peculiar sensation is carried to the palpating hand; this is known as tussic fremitus. If he is suffering from much secretion in the bronchial tubes or a bronchitis, then the respired air passing through the tubes will produce a murmur which is imparted to the palpating hand and this is described as rhonchial or bronchial fremitus. In practicing palpation it is most important always to remember that the palpating hand must lie firmly against the chest wall of the person to be palpated.

Attention should here be directed to the fact that in muscle spasm demonstrable at the apices that the upper border of the trapezius extending from the occiput down to the shoulder feels thicker, more spastic to the fingers, and that in grasping the thickened upper border between the thumb and the index or middle finger, a perceptibly larger amount of muscle tissue is noticeable as compared with the opposite or still unaffected side. It seems as if the palpating fingers pick up a greater volume of muscle tissue and integument in a given time.

CHAPTER 15

PERCUSSION

Percussion as practiced as an aid to physical diagnosis consists in gently, striking or tapping the chest wall, either directly or indirectly, by means of the tips of the fingers, with a force sufficient to throw into vibration the underlying structure to be examined. The method of percussion as applied in the examination of the chest is of comparatively recent adaptation, and, although percussion had been practiced in various industries for many years, as, for instance, the carpenter percussing the wall with a hammer in looking for a suitable place to drive a nail, etc., it was not until 1761, that Leopold Auengrubber of Vienna, in a classic description, first clearly defined this method of physical examination. With the aid of percussion it is possible to outline perfectly, on the outer chest wall, the apices of the lungs and the heart, the large blood vessels and the anatomical borders of the healthy, as well as the diseased lung, and by the nature of the sound elicited to determine the density of the parts percussed. (2) (4)

Topography of the Thoracic Organs. (31) A study of pulmonary tuberculosis or of the general pathology of the lungs presupposes a thorough knowledge of the position of the normal lungs within the chest cavity, and the ability to outline on the thoracic wall the various parts of the lungs, the position of the heart and its borders, the divisions and the borders of the lungs on inspiration, on expiration, etc. In order to simplify this study, the chest is primarily divided into the anterior, the posteror, and the two lateral surfaces, each again being arbitrarily subdivided in order to facilitate memorizing of the parts and convenience of study.

The anterior surface of the chest wall is divided and subdivided by a series of lines from above downwards, intersected at right angles from side to side. A line drawn from the suprasternal notch over the center of the sternum down to the ensiform cartilage and beyond is known as the midsternal line. This line divides the chest into equal halves, the right and the left.

A similar line drawn parallel and following the junction of the ribs with the sternum is designated as the costo-sternal line, and another line from the middle of the clavicle down to the lower costal border and through the nipple is generally known as the mid-clavicular, mammary, or nipple line, an outer line dropped from the shoulder down to the lower margin of the ribs, parallel

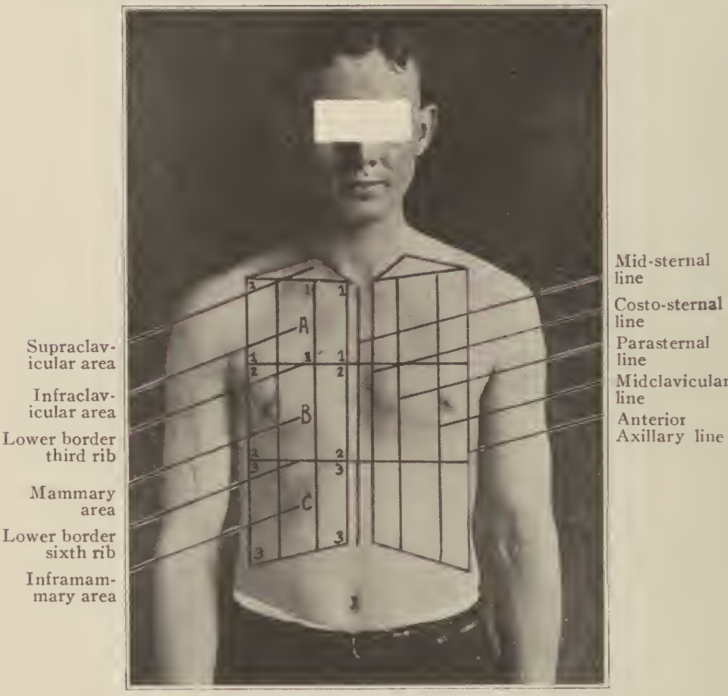


Fig. 12. The topographic area of the thorax (Anterior). The areas of the lungs as outlined on the anterior chest—right and left.

A. 1. 1. 1. 1. Infraclavicular area. The clavicle above, third rib below, costo-sternal line inner and anterior axillary line outer.

B. 2. 2. 2. 2. Mammary area. Third rib above, sixth rib below, costo-sternal line inner and anterior axillary line outer.

C. 3. 3. 3. 3. Inframammary area. Sixth rib above, costal arch below, costo-sternal line inner and anterior axillary line outer.

to the nipple line, and about 10 cm. to the outer aspect is called the anterior axillary line, and a vertical line midway to the mammary or nipple line and the costo-sternal line, is spoken of as the para-sternal line. All these parallel lines are intersected at right angles by two horizontal ones, one on a level with the lower margin of the third rib, the other that of the sixth rib, and these two lines with the parallel mid-sternal and anterior axillary lines divide the anterior chest into six nearly equal divisions,

three on either side, a right and a left upper infraclavicular, a middle or mammary, and a lower or inframammary area.

The posterior surface is divided as follows: a vertical line along the center of the spine divides the posterior chest into two equal halves; this line is designated as the vertebral line. Another line drawn from the middle of the shoulder down and

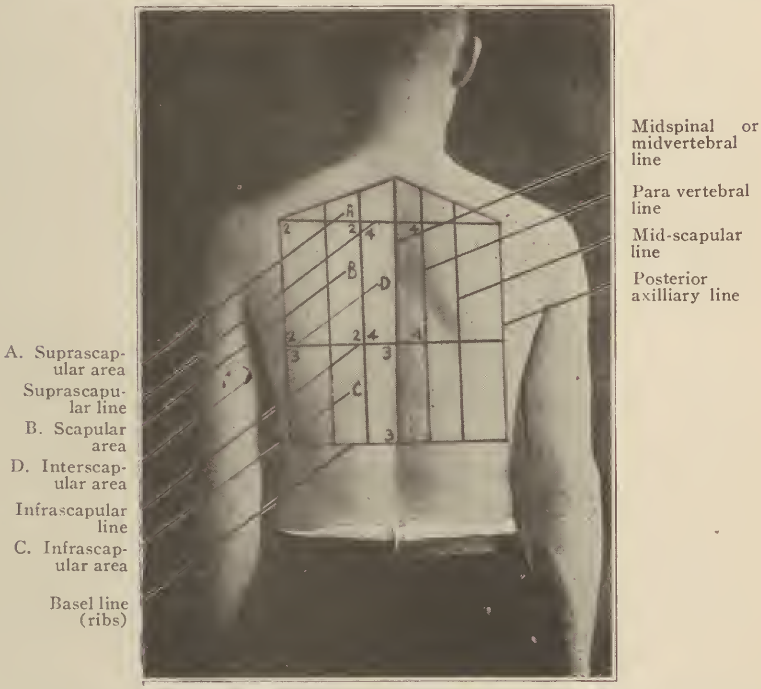


Fig. 13. The topographic area of the thorax, posterior. The area of the Lungs as outlined on the posterior chest, right and left.
B. 2. 2. 2. 2. The scapular area. From the spine of the scapula to the angle of the scapula and from the paravertebral line to the posterior axillary line.
C. 3. 3. 3. The infrascapular area. From the angle of the scapula to the lower border of the lungs and from the vertebral line to the posterior axillary line.
D. 4. 4. 4. 4. Thc interscapular area. From the line along the spines of the scapulae and the line horizontal to the angles of the scapulae and the paravertebral lines right and left, (or more correctly the area between the inner margins of both the right and left scapulae).

parallel to the vertebral, to the angle of the scapula, is known as the scapular line, a line from the shoulder along the axilla, the posterior axillary line, and a line midway and parallel to the vertebral and mid-scapular line, the paravertebral line. These posterior thoracic lines are intersected at right angles by an upper one along the spines of the scapulae, and a lower one on a level with the angles of the scapulae. These lines divide the

posterior surface of the chest also into six but unequal divisions, a right and left upper, or supra-spinous, a mid-scapular, and a lower or infrascapular. The area between the inner borders of the scapulae on either side and from about the first thoracic spine down to the seventh; or eighth is also known as the interscapular.

The lateral surfaces of the chest wall on each side are divided by a horizontal line along the lower border of the sixth rib, the anterior axillary line of the anterior division of the chest, and the posterior axillary line of the posterior division, forming the vertical boundary lines of this area; this area is again divided by a vertical line through the center, the "mid-axillary line." These lines divide the axillary area into the supra-axillary, the area above the sixth rib, a right and a left, and all that portion below this rib into the infra-axillary, also a right and a left.

The area over the sternum anteriorly is divided into the upper sternal, mid-sternal, and xiphoid, and by the mid-sternal line into a right and a left sternal.

The triangular area above the clavicle is known as the supra-clavicular, bounded by the clavicle below and inside by a line running upwards and outwards along the border of the sterno-cleido-mastoid muscle; the outer margain of this triangle is a line running from the middle of the clavicle upwards and inwards, meeting this first mentioned line about two inches above the clavicle. This area is occupied on either side by the apices of the lungs; the right apex is supposed to be slightly lower than the left, although this is considered pathological by some. An area on either side bounded above by the clavicle, below by the lower border of the third rib, internally by the costo-sternal line, and externally by the anterior axillary line is known as the infraclavicular area in which pure lung structure is found.

The area from the lower border of the third rib to the lower border of the sixth, and from the costo-sternal line to the anterior axillary line is the mammary area. This area on the right is covered by lung structure throughout, the dome of the liver extends into this area as high as the fourth rib, still covered by lung tissue, and on the left side in this area the heart lies superficially, being only partly covered by lung tissue. That part of this area between the heart and the anterior axillary line is covered by normal lung structure. The area below the sixth rib on either side and as far as the lower costal arch, is the inframammary; and here the right side is covered by the liver, very little if

any lung structure extending below the sixth rib, and on the left by the stomach, where Traube's crescent shaped space can be demonstrated. Posteriorly in the area above the spine of the scapula, the supra-scapular, over the scapulae, the scapular, be-

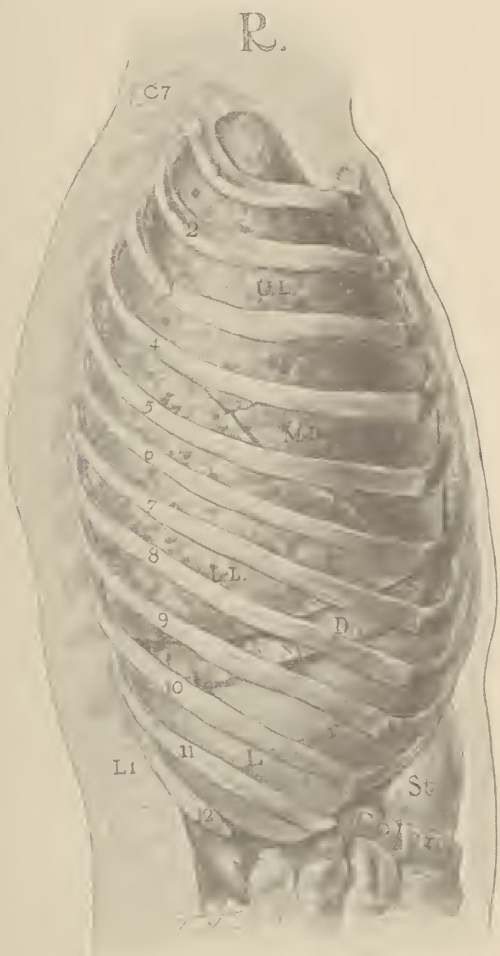


Fig. 14. The viscera of the thoracic cavity right side (R) in situ. Note that the apex of the right lung is conical.

tween the scapulae, the interscapular, and below the angle of the scapulae, the infra-scapular, and extending from the first dorsal spine above to the tenth rib below, is normal lung tissue covered by the bony and fleshy structures of the posterior thoracic wall. In the axillae both right and left that part known as the supra-

axillary above the sixth rib is covered by lung structure, and the area below the sixth rib on the right side by the liver, on the left by part of the stomach and the anterior portion of the spleen between the ninth and eleventh ribs, the posterior portion lying

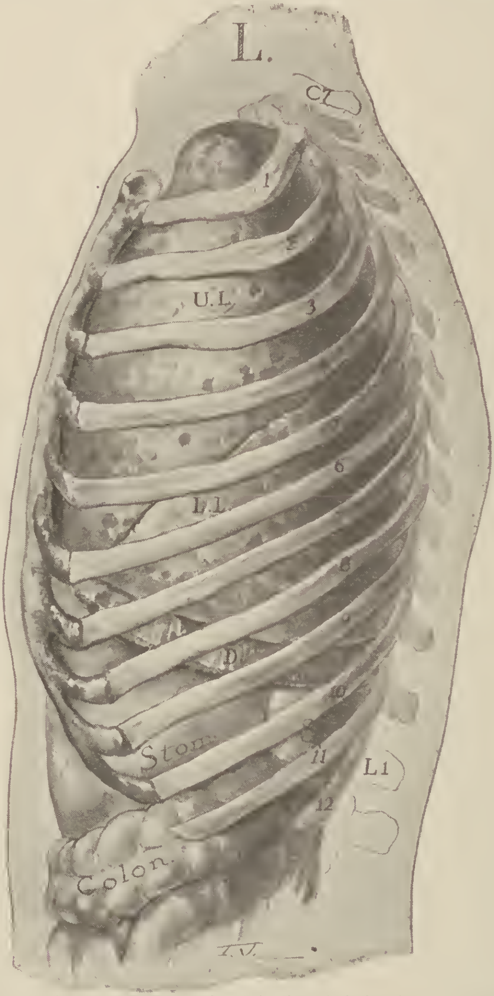


Fig. 15. The viscera of the thoracic cavity left side (L) in situ. Note that apex of the left lung is dome shaped.

in the left infra-scapular area. The lungs themselves, as stated above, are usually divided into the right and the left lobe, the right being again divided into an upper, middle and lower, the left into an upper and a lower. These divisions, dividing the lobes of the lungs into smaller lobes, three on the right half, and

two on the left, are known as the fissurae or incisurae, the incisurae interlobares, and they begin posteriorly on either side of the spine on a level with the cartilage between the second and third thoracic vertebrae, the right fissure or incisor running forward and downward in the right axillary area along the fourth rib, then dividing, the short branch following the course of the rib, terminating at the edge of the lung just behind the junction of the rib with the sternum, the other, the longer branch running downward, forward and inward, terminating opposite the midclavicular line, these fissures thus dividing the right lung into an upper, a middle, and a lower lobe; on the left side the fissure begins opposite the right, and running forward and obliquely downward, terminates near the lower border of the lung behind the parasternal line, dividing the left lung into an upper and a lower lobe. The lungs normally extend about an inch and a half to two inches above the clavicles, the left a trifle higher than the right, the lower margin extending in the mammary, or midclavicular line to the sixth rib in the midaxillary, to the eighth, and in the scapular to the tenth rib, the complemental pleural space about two inches below the margin of the lungs, and with the exception of a small portion of the left mammary area which is occupied by the heart, the lungs fill all this cavity, or at least the lungs lie in close contact with the entire surface of the thoracic wall.

The Borders of the Lungs. On the posterior chest wall the inner border of both the right and the left lung extend parallel on either side of the spine from the tenth rib upwards to the second thoracic vertebra, from which points they deviate, running upwards and outwards, reaching the highest point on a line opposite the lower margin of the vertebra prominence. The anterior borders of the lungs in the supraclavicular area correspond closely to the outer margin of the sterno-cleido-mastoid, on either side extending downwards and inwards, converging to a point opposite Ludwig's angle or a trifle below, whence they run parallel to the fourth rib, the right continuing down to the sixth and joining the lower border on a level with the midclavicular line, the left border at the fourth rib running outward and downwards, leaving the heart exposed, then turning inwards and downwards, joining the lower margin on a level with the left mid-clavicular line. This lapet of lung structure at this point is described as the Lingula Pulmonalis and the depression or

hollow where the heart is free is known as the cardiac depression or hollow indentation. (Incisura Cardiaca.)

Methods of Percussion. As in all other methods of chest examination, so also while practicing percussion the patient must be in an easy and comfortable position. The anterior chest wall is best percussed with the patient in a sitting or standing position, the arms hanging leisurely by the side of the body; for posterior percussion the patient either sits or stands, with arms folded over the chest or resting in the lap, the body leaning slightly forward; for lateral percussion the patient's arms should be resting upon the head.

Percussion is usually practiced by gently striking or tapping the thoracic wall with the tip or tips of the fingers, at the same time listening carefully to the sound produced, and noting the resistance offered to the percussing fingers. If the chest wall is tapped directly with the finger, like in striking the clavicle with the tip of the finger, it is known as immediate percussion; if, however, something is placed between the chest and the percussing finger, and the blow directed upon this, it is known as mediate percussion. Immediate percussion is now only applied when percussing the clavicles; over all other portions of the chest mediate percussion is practiced. The usual instruments necessary for mediate percussion are the plexor or hammer, and pleximeter or anvil; however, the best and simplest means always at hand, is to make use of the middle finger of the left hand placed in close contact with the thoracic wall as a pleximeter, using the middle finger of the right hand bent at right angle at the second joint as the plexor or hammer. Some examiners use the index, middle, or ring finger, or may use two or three fingers jointly as a plexor.

In beginning to percuss, the student should always remember to percuss very lightly at first, to practice light percussion at all times; heavy percussion is needed only if the area to be percussed and to be vibrated is deep seated, or we have reason to suspect that healthy lung tissue overlies an infiltrated area which we wish to throw into vibration. If a small infiltrated area should lie near the surface and much healthy lung tissue below this, then heavy percussion would vibrate this healthy tissue, and we could not gain a clear conception of the tissue overlying the healthy lung. Percuss lightly and with a force sufficient to vibrate the underlying tissue at a depth and a circumference of

about 5 to 8 cm.= 2 or 3 inches, and if in doubt about the area being more deeply involved, resort to heavy percussion. On the average, we find that most physicians percuss too heavily, but long experience in teaching has convinced me that the proper way of percussing can only be acquired by long practice, like all the other methods of physical examination. It is most important here to again remind the student in physical diagnosis that in the science of percussion it is necessary to carefully note the sound elicited, as to its pitch, quality, duration, etc., and the amount of resistance offered to the percussing finger.

Percussion of the Normal Chest. It might not have been necessary to have gone into details of how to percuss, as the technic is supposed to have been sufficiently mastered by all, while students in physical diagnosis. The object of giving the method of percussion here at some length is intended simply to refresh the memory of the student or physician who is doing tuberculosis work, to again remind him of the conditions of these sounds which he is expected to find over normal lung tissue, and to enable him to compare these sounds and the resistance, if any, with the sounds produced by the altered condition of the lungs, the tuberculous infiltrate.

Always begin percussion on the anterior chest wall, left side, just below the clavicle, in the mid-clavicular line. The note which is there elicited on percussion is called the normal resonant; it is soft and low in pitch, accompanied by a certain degree of resiliency. It is the given individual's most resonant note, more so at this point than over any other part of the thoracic wall. This normal resonant note obtained below the left clavicle should at once be compared with the note obtained over the corresponding area below the right clavicle, and the difference noted; here the note although resonant is normally found somewhat higher in pitch, and slightly more resistant. Next by immediate percussion tap the left clavicle above this point and at once compare it with a similar point on the right clavicle, and again note the slight difference in pitch and resistance and now percuss the lung apices above the clavicles, first the left apex, and then the right, and again note the normal differences. Next percuss the left chest below the clavicle down to the third rib, mid-clavicular line, and then compare it with its mate the right. Throughout the whole upper portion of the left lung and down as far as the third rib this normal resonant note will appear soft,

and the tissue slightly more elastic to the touch, than that of the right. As each individual has his or her own special note, we cannot know in advance what that normal note is, nor can this note be described, but must be learned in each new case by comparing the left most normal with the right less normal area. Each individual has a resonant percussion note distinct and definite. A percussion note of the greatest acuity can be elicited in the spaces between the heads of the sterno-cleido-mastoid muscles, just above the sternal end of the clavicles. Here percussion must be practiced in the direction of the lungs, and not towards the trachea. Insert the tip of the middle finger of the left hand between the heads of the muscles, the finger being bent at right angle at the second joint, as a pleximeter, and with the middle finger of the right hand as a plexor, percuss upon the knuckle of the inserted finger, keeping the finger well in the direction of either the right or left shoulder. The slightest difference in the percussion note, here directly upon the lung tissue is then very readily discernible. The normal difference in the percussion note of the left and right sides, as well as the normal difference of the same side, may readily be observed, next by percussing first on one side and then on the other, beginning at the median lung border below the clavicle near the sternum and percussing outward and towards the shoulder along a line below the clavicle. A perceptible but normal difference is then observed, being higher towards the shoulder but lower towards the sternum. Percussion sounds may be much enhanced by placing the patient whose chest we desire to percuss with his back against a door, hollow wall, or anything which may act as a sounding board, when by fairly heavy percussion the difference between the two sides is distinctly noticeable.

After having percussed the left apex anteriorly and having compared the note obtained with that of its mate on the opposite side, the right apex, percuss the left lung from the apex down to the lower margin about to the sixth rib, first along the mid-clavicular line, and then along a line midway between this and the anterior axillary line. In the mid-clavicular line a change of note is observed below the fourth rib, where the heart is first encountered deeply situated, the note still having a resonant quality but becoming more and more dull with continuous downward percussion, and gradually completely flat where the heart lies superficially and as the area over the heart is passed, the note

again changes now to tympany, indicating the location of the stomach. The percussion note from the apex of the left lung down to the lower border along the line parallel to the anterior axillary line is usually clear and resonant, and though slightly modified by the proximity of the heart and the stomach below, and over the right half of the chest anteriorly, as already has been stated, the note is slightly higher, on percussion from above down, when at the fourth rib, the note changes, becoming higher on fairly firm percussion, where the dome of the liver lies, deep and continuing percussion downward the note gradually blends into flatness or complete absence of resonance over the liver at the lower margins of the lungs. We next percuss the posterior thorax, beginning at the upper part of the chest along the paravertebral line from above down to the lower margin of the lungs, usually on a level with the tenth rib, percussing first over the left, then over the right lobe, and here, as on the anterior chest wall, the difference over the two sides is again apparent, the note being more resonant, less high, softer, but resilient over the left and less so over the right, where it is slightly less resonant, somewhat higher, and less resilient.

In percussing the axillary area, the spleen is encountered at the left, between the ninth and eleventh ribs, extending anteriorly as far as the midaxillary line. Over this area the note is flat, but being resonant over all the other portion and over the right axillary area it is slightly higher, changing to flatness at the lower border where the liver lies superficially. In percussing the upper axillary area, high up in the axilla, the method in use on the anterior chest, as between the heads of the sterno-cleido-mastoid muscles, should be practiced, putting the tip of the middle finger of the left hand as a pleximeter well up into the axillary space. (44) Deep inspiration and full expiration greatly modify the percussion note, making it more resonant, even tympanitic on full inspiration, and correspondingly less resonant, slightly higher on expiration. The student should always bear in mind that in the normal over all that area over which lung tissue lies in close proximity to the walls of the inner thoracic cavity, a normal resonant percussion note can be elicited, modified by the overlying bony structure, by the sternum anteriorly, and the scapulae posteriorly and by the region over the heart, liver and spleen. He should also remember that this percussion note finds its most normal and natural expression over the apex of the left

lung, particularly anteriorly below the clavicle, that over the right apex a mixture of resonance with dulness can always be elicited as normal, that the excursion and aeration of the upper apices of the lungs, especially the right, is usually very slight, that in the respiratory movements the apices expand only a few millimetres, that the excursion is mainly in a lateral and anterior direction, and that it is over this area that the percussion note can best be studied; and that posteriorly in the adult the lung extends upward on a level with the spinous process of the first dorsal vertebrae but that in the newly born the apices lie somewhat lower, more on a level with the tracheo-bronchial lymph glands.

Percussion of the Abnormal Chest. A tuberculous process consists either in the filling up of the alveolar spaces with an inflammatory exudate, as in beginning or acute disease, or in fibroid changes gradually taking place in the pulmonary tissue, as in the chronic form. This lessens the normal air content of the lungs, in consequence of which the normal resonant, low, soft, full percussion note becomes less intense, but shorter and higher in pitch. If the tuberculous process is not extensive, as is frequently the case, the surrounding healthy lung tissue may become collapsed and be then accompanied by a tympanitic quality of the percussion note, tympany being higher in proportion to the size of the collapsed area, but if the tuberculous process is extensive, then the percussion note becomes more or less dull, and in proportion to the extent and the nature of the diseased process, all gradations from normal to tympanitic and from dull to even flat can be demonstrated. It is evident from this that especial importance must be placed on the early recognition of the primary changes in the note from the normal.

On the anterior chest begin percussion, as has been stated, on the left side below the clavicle, mid-clavicular line, making a few soft taps and then compare it with the opposite right side over a corresponding area. If the note over these areas is alike in resonance on both sides, or perhaps the left a trifle higher, then we have fairly positive evidence that the left apex is infiltrated. It is understood that we consider the normal differences in the muscular development in left handed people, etc. To compare slight differences between the two sides, percuss first the left side by making but a single tap, using fairly firm percussion, and then percuss immediately a similar area over the right chest,

or percuss directly upon the clavicle, first upon the left, then the right. This single tap percussion should be repeated quite quickly from left to right, like, one two, one two, one two, etc., making about six to ten single taps in quick succession on both sides, the slightest difference between the two sides being readily recognized in this way. This is far superior to the method of making six or eight strokes first upon one side, then upon the other, at the same time trying to carry in your mind the note observed first upon the left, then upon the right. Having found here a difference in the percussion note, as compared with the normal, next percuss from the inner margin of the lung, near the sternal end of the clavicle, towards Mohrenheim's fossa along the lower border of the clavicle, and observe the change in note. In the normal, the note changes as we percuss towards the shoulder, becoming less resonant, but at the sternal end a slight tympanitic quality accompanies the note. If. however, either lung is infiltrated, the note does not change, as we percuss from left to right or right to left. Percuss in the direction of the lungs not towards the trachea nor towards the shoulder. Having found an apical involvement, next percuss downwards in order to outline the extent of infiltration, and here again the percussion note will change from dull to resonant and resiliency, as we approach normal lung structure. If only one apex is involved, then percuss this apex, first on full inspiration, while the patient takes a deep breath, and still holds the breath, then on full expiration, the patient again holding the breath, and compare this with the note obtained over the healthy side under similar conditions. Over the healthy side a decided difference in the percussion note on deep inspiration and full expiration is most distinctly noticeable, the note becoming higher with full expiration, and more resonant, even tympanitic on inspiration, but no such change will be observed over the infiltrated area.[1]

If a less amount of air enters the lungs on inspiration than in the normal respiratory act, the difference in the percussion note between that obtained on deep inspiration and full expiration may be very little, or may be entirely lost, or if but very little air enters on inspiration, then the difference between the inspiratory and the expiratory percussion

[1]The difference in the percussion note over various parts of the chest can more readily be noted and studied by means of stethoscopic percussions. Place the bell of the stethoscope anteriorly on the sternum or posteriorly over the spine and note carefully the sound produced while percussing different areas of the chest. This is best done while one or more students are listening, the instructor doing the percussion. For instance, first percuss the left and then the right clavicle, next the area below the clavicles, mid-clavicular line, then percuss any part of the neck lateral or posterior, or perhaps the shoulder, then the chest over normal lung structure, and the difference in the percussion note between high and low will become readily apparent.

note may be very faint, usually there being no difference at all. Frequently in beginning apical involvement the percussion note during inspiration is low, and high during expiration. On very light percussion, however, we usually find the reverse, the percussion note being higher on inspiration.

The difference in the percussion note, when one apex is infiltrated and the other is still free from the disease, can be more distinctly elicited as has been stated previously if the patient's chest is percussed while his back is firmly placed against a panel door, hollow wall, or box to act as a sounding board. In be-

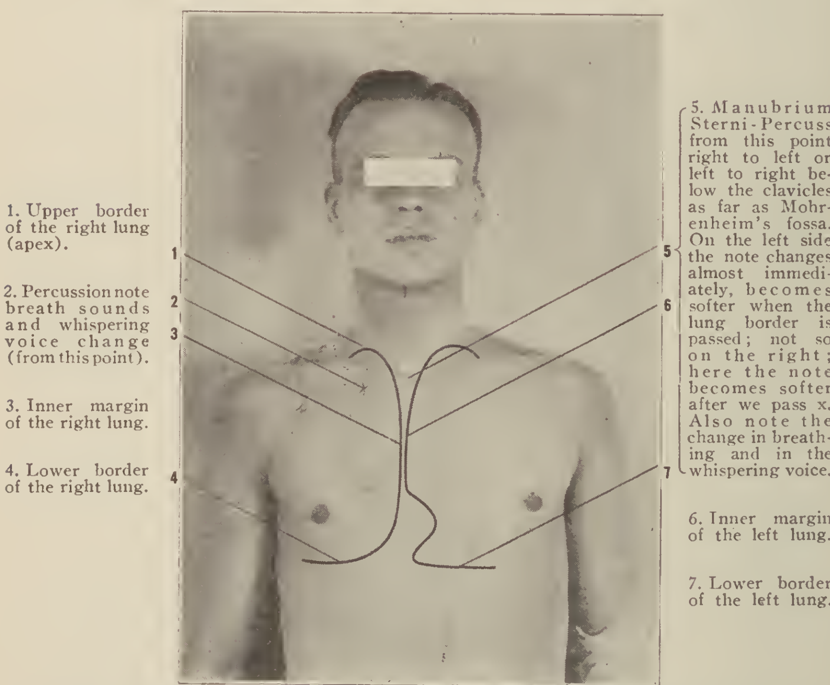
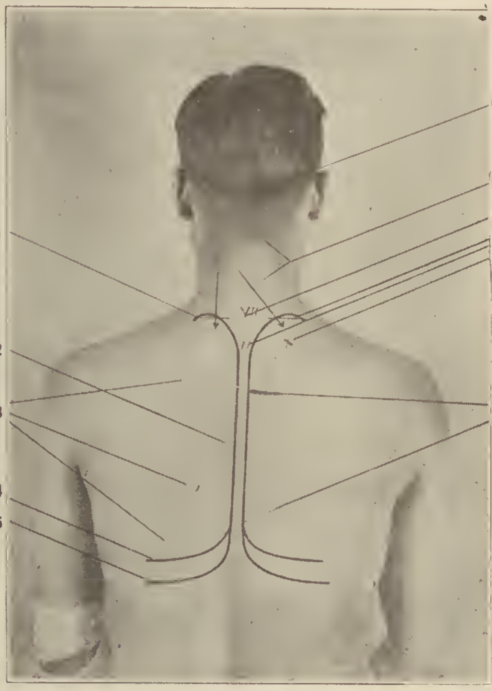


Fig. 16. The normal Lung borders of the anterior chest. Showing the normal variations in the percussion note, the stethoscopic changes in the breath sounds and in the whispering voice (normal).

ginning apical disease, if the area above the clavicle is percussed, the lower position of the apex and the lessened amount of lung volume on the affected side is also readily demonstrable. Next percuss the apices posteriorly and compare the infiltrated side with the normal, remembering always the normal difference between the right and left side, and as over the anterior chest the diagnosis by percussion could most readily be made if the left

apex is primarily involved, so can also the posterior upper lobe, the left sided infiltration, be more easily recognized by percussion. The involvement of one or both apices may fairly well be outlined by the following method:

At the second thoracic vertebra, on either side, the inner margin of the lungs approach, and from this point run parallel along



1. Upper level of lung on a line with the cartilage below the 7th cervical vertebra.

2. Inner margin of left lung.

3. Area of normal, vocal or voice resonance.

4. Lower border of the lung, ordinary breathing.

5. Lower border of the lung, deep breathing.

6. Pectoriloquy. Heard here frequently in some individuals perhaps somewhat distant and not very intense.

7. Bronchophony.

8. Vertebra Prominens.

9. Upper margin (apex) of the lung. Above this the breath sounds are bronchial.

10. Second dorsal spine.

11. X. Between this and the upper border of the lung breathing is broncho-vesicular.

12. Inner border of the right lung.

Fig. 17. The normal Lung Borders of the posterior chest. Showing the change of note in the normal when percussing from an airless to an air containing structure. This becomes still more definite when we auscultate the breath sounds and the whispering voice.

Arrow Left. Percuss paravertebral line—the note changes almost immediately when the upper margin of the lung has been passed. Also note the change in the breath sounds, particularly note the change in the whispering voice.

Arrow Right. Percuss diagonally. Here the note does not change as abruptly when the lung border is passed, it only becomes soft when we pass X. Here also note the gradual change in the breath sounds and the whispering voice.

the spine to the lower border at about the tenth rib. The upper border or highest point of the lung in the paravertebral line is on a level with the upper border of the cartilage, of the first dorsal vertebra. Here let us draw a horizontal line. If from the second dorsal vertebra a line is drawn upwards and outwards crossing the paravertebral on a level with the first dorsal vertebra and continuing upwards and outwards to the horizontal line

we will form a line which runs quite parallel with the upper lung border. If now we percuss above this line, we are percussing over airless tissue, over the neck, bone and muscles, and we elicit a note which is distinctly high pitched, short, very resistant, and flat. If we then percuss downwards a perceptable change is noticeable as we pass this line in the normal, the note becoming soft, low, accompanied by a palpable resiliency; however, if the area is infiltrated, there is only a slight or no change when we cross this line, depending mainly on whether the area is intensely consolidated, or if the involvement is but slight. Now, if a primary involvement should be below this line, say from three to five cm, the lung being still normal up to this line, then as we percuss from above down and approach this line, the

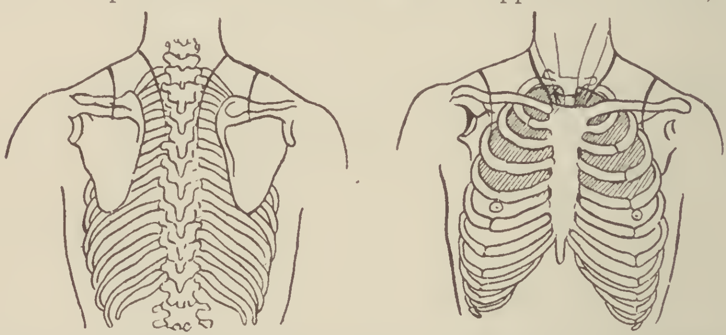


Fig. 18. Isthmus percussion in health according to Kroēnig (Schematic). Anterior and posterior view. (Normal.) The narrowest part is about 4 cm in width, equal on both sides. (Here a perceptible difference in one sided tuberculous infiltration is noticeable on percussion.)

note does not blend into the flat note, as it would if the extreme upper lung margin were involved, but we perceive a resonant and lower note, changing as we continue downward and reach the seat of infiltration, to high and short, its intensity depending, as in all pulmonary consolidation, upon complete or partial involvement. If in doubt, percuss first about two to four cm, above this line and then immediately over the supposed infiltrated area as a comparison, and the great similiarity of the percussion note in both will become apparent, perhaps slightly modified if the diseased process is not entirely airless. The note percussed above this line, as well as the note percussed over the supposedly infiltrated area, should next be compared with a normal lung area, so as to fix definitely in the mind of the student the difference between the normal flat note above this line, the abnormal flat note below this line, and the normal soft, resonant note audible over healthy lung structure. Next, should the flat note

above this line blend with a similar flat note below the line, then continue percussion in a downward direction until a decided change to resonance has been secured, and now draw a line at this point, and the extent of infiltration reaches from this point up to the line indicating the upper lung border, and now, on percussing this area in either direction from right to left, or left to right, the extent of the consolidation may be outlined. Next continue percussion in a downward direction along the paravertebral line to the base of the lungs. Percussion of the lower lung borders should never be omitted, although in by far the greater number, the primary tuberculous process in adults is in one or both apices; however, tuberculosis of the lower lobes as a beginning disease is sometimes encountered. This is particularly so in children and occasionally in adults. It is also of importance to remember in this connection that in individuals suffering from diabetes, if complicated with pulmonary tuberculosis, the primary process in the lung is often found in the lower portion of the upper lobe and not in the apices of the right, middle nor in the upper portion of the lower lobe. In patients suffering from chest deformities, the primary process is usually in the lower or middle right lobe. One must remember, however, that notwithstanding much thoracic deformity, such as scoleosis, kyphosis, etc., that in children, as well as adults afflicted with such deformities pulmonary tuberculosis is very infrequent.

The upper lung structure about the shoulder, known as the isthmus of the lungs, can also, according to the methods of Kroenig and Goldscheider, (44) be distinctly outlined, and percussion should be here of the mildest type, the sound produced by the percussing finger should scarcely be audible, and the resistance and the faint note should be observed particularly. In pulmonary tuberculosis, owing to early contraction of the pulmonary tissue, the isthmus over the affected side is less in extent, from 1 to 2 cm, depending upon the tissue involved. In the normal, both sides are about alike, varying in different individuals, from 5 to 6, and more cm. The isthmus, or the narrowest part of the lungs can be outlined here on the anterior as well as the posterior wall of the chest.

In practicing percussion, the axillary area, especially the upper, should never be omitted and with the patient's arm resting well on the head, the tip of the examiner's finger should be placed way up in the axillary groove as a pleximeter.

Aberrations. The note on percussion may frequently be complicated by various aberrations, to which attention may be directed, as for instance, by emphysema, by pleurisy, by cavities, etc. If emphysema surrounds a tuberculous area, the note then will be low, somewhat hyperresonant, or, as frequently happens, if a whole lobe becomes tuberculous, the remaining healthy side will do compensatory work, giving a more or less tympanitic note. If a pleurisy accompanies the tuberculous process, the acute onset, the pain, the flat note, evidence of a compressed lung above the fluid, can be utilized in making the diagnosis, and when cavity formation has taken place the evidence of vomicae is easily demonstrable by percussion, the difference in the percussion note with the mouth of the patient open and closed being often readily demonstrated. Wintrich's note in the presence of a cavity, the patient breathing in and out with the mouth wide open, the percussion note will be high and loud, becoming low and soft with the mouth closed and breathing in and out through the nose. Cavities are usually sharply defined and outlined over small areas. Cracked pot sounds are best demonstrable while percussing quickly and heavily, the patient keeping the mouth wide open. A natural pneumothorax occasionally accompanies an active case of pulmonary tuberculosis, particularly if one side is extensively involved. This and a band-box percussion note, great resistance to the percussing finger due to the compressed air in the chest cavity, are evidence of a collapsed lung. Immobility of the diaphragm on the affected side, (William's Symptom) should be mentioned, being higher on the diseased, as compared with the healthy side, and may aid in the diagnosis of tuberculosis by the method of percussion.

Sources of Error. Percussion sounds which may be mistaken for tuberculous infiltration. Over different areas of the thoracic cavity over which we expect to find a normal note, on percussion we may at times find some changes, and this may lead to wrong deductions, unless we bear in mind that something other than a pulmonary tuberculous process may admit a change in the percussion note, which may often resemble that of tuberculosis. This particularly is to be borne in mind when percussing the chest wall in children. Error in percussion may also be due to a faulty technic, or to the variations in the anatomical relationship existing between the two sides, hence:

(1) Attention should be directed to the difference in the mus-

cular development on the two sides, the somewhat greater muscular development in right handed or left handed people, all of which may greatly influence the note being higher over the thicker muscle.

(2) In scoleosis, kyphosis, etc., due to the contour of the spine, the note is higher over the muscle on the bulging side and lower over the retracted or atrophied area.

(3) In percussing the clavicles too near the sternal end, too far inward where a tracheal note is added, or again too far outwards, towards the shoulder and beyond lung tissue, the note may be flat.

(4) Percussing over enlarged bronchial glands, on either side of the spine and upwards towards the apices a dull note is elicited which may be mistaken for pulmonary involvement. A similar note is frequently noticed anteriorly in the parasternal line right side in the second and third interspaces.

(5) In children a collapse induration of the lung, as first pointed out by Kroenig, due to insufficient aeration and usually, the result of nasal or mouth obstruction, adenoid vegetations, hypertrophic turbinates, tonsillar hypertrophy, etc., may give signs which simulate infiltration. That this is not due to tuberculosis the clinical picture can prove and besides, the student must remember that pulmonary tuberculosis in children is comparatively infrequent. This condition is also occasionally found in adults as the result of early nasal trouble.

(6) Chronic bronchial inflammation, particularly bronchiectasis in children, due to compression of the upper lung, may give rise to consolidation, and on percussion give a high pitched resistant note, not due to tuberculosis.

(7) In pneumoconiosis, siderosis, chalicosis, etc., the percussion note is more diffuse than in pulmonary tuberculosis, due to early retraction but without destruction of lung tissue.

(8) Neoplasms, carcinoma, sarcoma, syphilis, actinomycosis, etc., may all, if involving the lung, give a percussion note which may simulate that of pulmonary tuberculosis, but the general history of the case and the cause of the disease will make the differential diagnosis definitely.

(9) Apical bronchitis, a chronic pneumonic process, etc., may all modify the percussion note from the normal, and it should also be remembered that apical percussion dulness has been occasionally observed in perfectly healthy lung tissue and confirmed by autopsy findings.

Palpatory Percussion. The subject of percussion can not be passed, would be but incomplete, if reference were not made to Palpatory Percussion. Notwithstanding the fact that a most able writer on physical diagnosis refers to it in very slight terms as of "very little value in diagnosis," if properly understood and carefully practiced it becomes one of the most valuable aids in our armamentarium. Palpatory percussion is practiced by making a few firm taps directly over various parts of the chest wall with the tips of the four fingers of either the right or left hand in quick succession, from the wrist and not from the elbow or shoulder, observing the note produced and the resistance offered. Chronic pneumonic processes, old pleural thickenings, muscular spasticity and atrophy, empyaema, pleurisy with effusion, emphysema, intra-pulmonary thickening due to neoplasms, etc., can most readily and quickly be discerned by palpatory percussion. It may, as compared with the ordinary method of percussion, be considered a crude method of diagnosis; it remains, nevertheless, if faithfully practiced and thoroughly mastered, a most valuable aid, and the student in physical diagnosis is well rewarded, and the time well spent for its mastery, continuous practice alone leading to its thorough comprehension. One must remember that, as in all other methods of physical examination, only continuous and faithful practice and repetition will lead to a thorough understanding.

THE (TUBERCULOUSLY) INFILTRATED LUNG. (SCHEMATIC.)

Explanatory remarks concerning the infiltrated pulmonary tissue, as is indicated by the shaded areas in Figures 19 and 20.

A study of the tuberculous lung by means of the physical signs of (1) Percussion; (2) Auscultation and (3) Whispering Bronchophony.

(1) **Percussion:** Begin percussion at A (Fig. 19) along the shoulder where the note usually is dull or flat; as we approach the upper, lateral border of the lung the note begins to change and as we pass the border the note becomes distinctly resonant; at the same time a resiliency is noticeable. This change would be still more in evidence if we percussed along the line B, and the pulmonary tissue were normal. Here, in Figure 19, however, the apex appears pathologic, and as we approach the lung border the note does not change to resonant as in the normal, but remains dull and continues so till we pass the dark area which in the Figure indicates the region over the second or third rib and interspaces, below which the note again becomes distinctly resonant. On the opposite side, the right lung, the extreme apex is still free, but an area of infiltration is indicated by the dark area towards the shoulder, extending, perhaps, from the second to the fourth rib. Now, if we percuss at C or, better perhaps, about 6 cm towards the right and percuss downwards, the note will

change from dull to resonant as we pass the upper lung border, differing greatly in this respect from the note elicited over the opposite, the left, apex. If next we percuss in the direction of D, we shall again notice that the percussion note does not change; we pass the lateral border of the lung as we find in the normal or as we found when percussing the opposite lung or from A down and inward but the note only begins to change from dull to resonant when we have passed the darkened, the infiltrated area and are again percussing over normal lung structure, where the note again becomes resonant and more resilient to the palpating finger. This from the shaded area in the figure would be at about the fourth rib and interspace. Again percuss at B and downwards, approximately following the left paravertebral line to the fourth or fifth rib, and then along a similar line on the opposite side (right paravertebral line), and the difference in the percussion note can be distinctly and clearly noted. Along the right paravertebral line the note changes almost abruptly as we pass from neck percussion, which is dull or flat, over to percussion of the normal lung area, that is, when we have passed the upper lung border, but not so when we percuss the corresponding area on the left side. Here the note does not change when we pass the left upper lung border, only when we have passed the shaded area does the note become again resonant. This same applies to Fig. 20 where the letters E, F, G and H indicate the direction of percussion over healthy tissue or from an infiltrated area through to normal lung structure. Here the changes in the percussion note are not so clearly demonstrable, that is, in comparing the area of the anterior with that of the posterior chest, excepting in the parts below the clavicles. Over the areas below the clavicles, begin percussion at the middle of the manubrium at X, percuss in a lateral direction following the arrows, from the sternum towards the shoulder. Over the left, the most normal side, where the tissue below the clavicle is still in great part normal, the note does not differ much from the full normal (here the proximity of the infiltrated to the normal lung changes the percussion note but slightly), the normal being very resonant in the mid-clavicular line, slightly so towards the shoulder, higher over the sternum. (For description, see text above, (this chapter), where the normal percussion note elicited over both right and left areas below the clavicles is given in full detail.)

(2) Auscultation. Placing the bell of the stethoscope anywhere along the neck, we usually hear a coarse sound, described as tracheal breathing. This sound in the normal continues until we pass the margin of the lung (left apex), when tracheal or bronchial sound ceases, to be displaced by soft vesicular breathing. Here, in Figure 19 at B, as we pass the lung border (left apex) the breath sounds do not change (do not become soft but remain harsh), but owing to tuberculous infiltration continue harsh till we have passed the darkened area, where at about the third interspace the breath sounds gradually change from the coarse inspiratory and prolonged expiratory to the soft inspiratory and still softer and shorter expiratory sounds. The same difference in the breath sounds can be demonstrated when we move the bell of the stethoscope in the direction of the arrow D. At C, however, the normal sounds become somewhat more apparent when we listen a little more to the right in the direction

of the right paravertebral line. This also applies to Figure 20 in the directions of the arrows E, F, G, H. Now place the bell of the stethoscope over the middle of the manubrium at X, where tubular breathing is distinctly heard. Next place the bell a few centimeters towards the left, and we note that the breath sounds begin to change, and as we pass the lung margin, over that portion where the lung is still free from tuberculosis we hear a distinctly vesicular sound, soft though continued with less intensity, perhaps slightly harsher in the infraclavicular fossa. Having studied the left area below the clavicle and having interpreted the sounds, we place the bell of the stethoscope for a second time at X, gradually carrying it towards the right lung border, in the direction of the arrows,

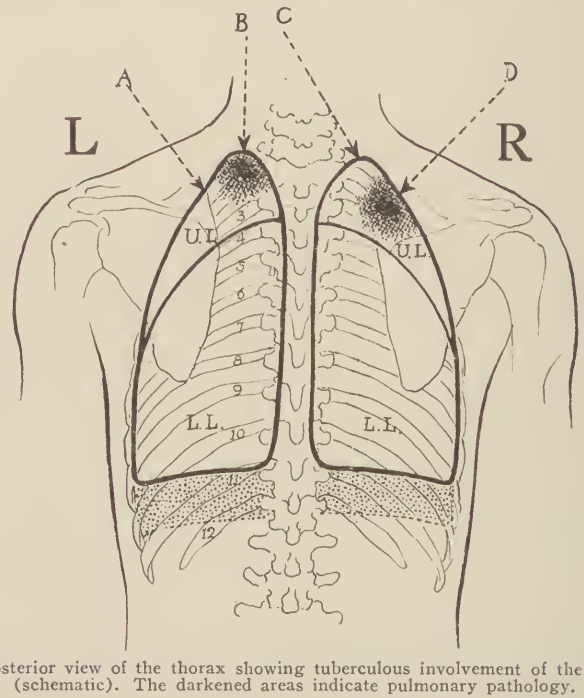


Fig. 19. Posterior view of the thorax showing tuberculous involvement of the upper lobes (schematic). The darkened areas indicate pulmonary pathology.

and we note that the sounds here differ much from the sound which we heard while listening along the lower border of the left clavicle. Here bronchial breathing is distinct at X and continues so as we proceed to the right, the breath sounds do not change as we approach the shaded area, there is perhaps a greater accentuation as we enter the densely infiltrated area. (Compare these findings with the breath sounds in the normal lung.) (This is fully described in Chapter 16—Auscultation.)

(3) Whispering Bronchophony. The Whispering Voice. Again place the bell of the stethoscope at B along the neck (Fig. 19) and follow the arrow in the downward direction, note that the whispering voice is distinctly transmitted to the ear. This in the normal should cease when we pass the lung border, the voice being heard but faintly or not at all. Here,

however, as we pass the lung margin the voice sounds are carried with the same intensity over this border and only begin to change when the darkened or infiltrated zone has been passed, where again the intense whispering sound stops almost abruptly, to pass into a faintly audible sound (or perhaps no sound at all). A similar study can be made at D, when following the direction of the arrow; here, also, the whispering voice changes when the darkened area has been passed, this at about the fourth interspace, paravertebral line. At A and at C a few centimeters to the right along the paravertebral line, the faintly, indistinct, or absent whispering sounds over normal lung structure can readily be studied so

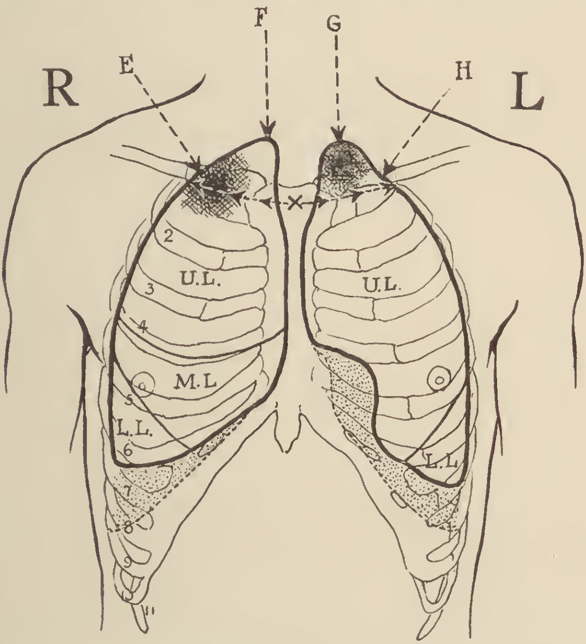


Fig. 20. Anterior view of the chest showing tuberculous involvement of both apices (schematic). The darkened areas represent the tuberculous pathology.

as to differentiate them from the pathological whispering sounds heard in the direction of arrows B and D. Next place the stethoscope on the anterior chest over the middle of the manubrium at X (Fig. 20) and carefully note the whispering voice as heard there, and gradually carry the bell first from X to the left shoulder and then from X to the right and note the difference in intensity of the whispering voice as heard toward the right infraclavicular fossa, where in the normal this sound should not be heard at all. (See Auscultation, page 157.)

The student's attention is specifically directed to these figures. The darkened zones here indicate a tuberculous infiltration, schematically. A tuberculous involvement of any part of a lung, be it at the apices, at the bases or in the middle lobe, can be outlined by percussion, auscultation and by the whispering voice and will conform with the changes as given in these schematic pictures.

CHAPTER 16

AUSCULTATION

(Including the Mechanism of Breathing)

General Considerations. After having made a thorough inspection of the chest, followed by careful palpation and percussion, we must then be in a position to state definitely whether the findings are those of a healthy or a diseased individual, whether they show a strictly normal or a pathological lung, before we resort to auscultation. Inspection, palpation and percussion have shown that changes have taken place in the underlying lung structure, but auscultation tells us what these pathological processes are, the kind and nature of these changes and whether these processes are active, arrested or healed. In a true sense auscultation is employed simply to confirm our previous findings, to clinch the diagnosis, and it should not be used, as it so frequently is in routine examinations, as the chief or sole method of physical examinations, but only as a confirmatory aid.

Auscultation as an aid in the early diagnosis of pulmonary tuberculosis depends upon the initial pathologic changes going on about the alveoli, alveolar ducts and the bronchiols. As this is usually accompanied by a more or less exudative infiltrate and as by means of these changes which are generally of a catarrhal nature, the free access of air to the periphery of the lung meets with an obstruction, followed by productive foreign sounds, such as are never found in the normal, and as the initial tuberculous process gradually extends and advances into neighboring and contiguous tissue, becoming more and more definitely catarrhal, primarily normal lung sounds give way to these changes, and from the vesicular one passes to the impure vesicular, to the bronchial and ultimately to the cavernous, accompanied at first by very fine but numerous crackling sounds, then more coarse, less numerous, more mucous and finely to amphoric, presenting a continuously changing picture.

Methods of Auscultation. Auscultation is defined (31) as the method of listening over the surface of the thoracic cavity to the

sounds produced within the chest wall during both the phase of inspiration and expiration. If in examining the chest of a patient the physician places his ear directly upon the chest wall while the patient breathes or speaks, it is known as immediate auscultation; if, however, the sounds produced within the chest are studied by means of an instrument known as the stethoscope, and this is the method now almost exclusively in use, it is known as mediate auscultation, that is, a medium is placed between the patient's chest and the ear of the examining physician. Auscultation or proper mediate auscultation was introduced to the profession by Laènnec, Renè Theophile Hyacinthe, a most distinguished French physician, about 100 years ago, namely in 1821, since which time it has been constantly in use. To mention the various instruments recommended for making auscultory examinations does not properly fall within the province of this work. Suffice to state, however, that every student should select the make of stethoscope most suitable to his hearing, because a certain instrument may be well adapted to the hearing of one while entirely useless to another.

Auscultation of the Normal Thoracic Sounds

The Normal Breath Sounds. The Normal Auscultatory Sounds. If we note carefully the respiratory movements of a healthy adult while asleep, we will observe that he breathes from 18 to 20 times per minute or that he performs that many cycles of breathing, and we will note further that each cycle of breathing is divisible into three distinct phases, the phase of inspiration, of expiration, and of rest, and that the phase of rest is equal to and even a trifle longer than both inspiration and expiration. With advancing years a slight change in each cycle is noticeable; this is the addition of a fourth phase, a short period of rest between inspiration and expiration. In the infant we often observe in the normal only two phases—inspiratory and expiratory.

If we place the bell of the stethoscope above the upper margin of the lung border, that is along the neck, either right or left side, and listen, we will hear a coarse inspiratory sound not unlike that of blowing through a tube, of high pitch, but no other sound; with expiration we will hear a nearly equal coarse but less intense expiratory sound a trifle longer than the inspiratory and a little higher in pitch; this is next followed by a period of rest during which no sound whatever is heard. This

is then immediately followed by another cycle of equal time and duration as the preceding one and so on.

These sounds which we hear at these points over various parts of the neck are designated as tracheal breathing but may also be described as intense tubular or bronchial, being produced in the largest bronchial tubes. Similar sounds, though of less intensity, are heard above the clavicles, over lung structure on the upper portion of the sternum, and in children may frequently be heard along either side of the spine as far down as the fifth or sixth dorsal vertebrae, but usually bronchial sounds about the thoracic wall over normal lung tissue are heard over a very limited area and throughout the greater portion of the chest cavity we hear a soft inspiratory sound low in pitch, followed immediately by a still softer, still lower and shorter expiratory sound. The duration of these sounds is usually described as 4 to 1, the expiratory only one fourth as long as the inspiratory. This is usually known as the normal vesicular murmur. This sound or murmur has been likened to the rustling of the leaves in the forests by the winds, but to my mind the best simile in nature is the sound produced by the waves along the shores of a lake, the waters rushing shoreward with a coarse sound (active motion), flowing back (passive motion) with a softer, less audible and shorter, often scarcely heard sound. This is then followed by a period of quiesence or rest after which the next wave occurs and so on.

Pure normal vesicular breathing as a physical symptom is an expansion murmur of the healthy pulmonary air cells. It is heard wherever healthy lung tissue is found underlying the bony frame work of the chest wall and it predominates greatly. To a limited extent only do we find different sounds over this area and these different sounds are usually described either as bronchial or broncho-vesicular. These three different sounds are the sounds heard over a normal lung and all other sounds heard are abnormal or pathological. Even these normal sounds, if heard over portions of the chest wall over which they are usually not heard, must be considered as pathological. For instance, hearing the vesicular sound over the heart area is just as pathological as hearing the bronchial at the base of the lung. Over the anterior portion of the chest wall the normal vesicular murmur is heard from the clavicles to the lower border of the lung, and posterior from the apex to the base, modified over the area

where the heart is superficial, over the liver, and by the thickness of the chest wall, the muscular development, the fatty infiltration of the skin, etc.

As in percussion, so also in auscultation, we find the area below the left clavicle the most normal in each individual and here in the mid-clavicular line the vesicular murmur is normally soft, a harsher quality being added as we listen on the right corresponding area. A distinctly harsher quality is also heard even over the left side, as we place the stethoscope more towards the sternal end of the clavicle, but it appears softer if we listen below the clavicle and towards the shoulder. As already stated, over the right lung below the clavicle, the inspiratory sound is harsher than over the left lung, less vesicular, but the expiratory is still more harsh and less vesicular when compared with the left and is at the same time slightly prolonged and high. This is known as the broncho-vesicular murmur. It is a sound midway between the soft normal vesicular murmur and the presently to be described normal bronchial. Over the posterior chest wall the normal vesicular sound is also heard best over the left apex, a slightly harsher quality being added over the right.

Over the upper portion of the sternum, the upper portion of the spine, to a slight extent near the bifurcation of the trachea, the 3rd and 4th dorsal vertebrae and perhaps a little on either side, as well as above the clavicles, the predominating sound is distinctly tubular, a high and intense inspiratory, and a higher and more prolonged but less intense expiratory murmur; this is bronchial or tubular breathing. This sound particularly if heard over an area over which it is normally not heard, is a true indication of pulmonary infiltration. Bronchial breathing if heard over otherwise healthy lung structure indicates a compact infiltration of parts of the pulmonary tissue or the communication of an air containing hollow space with a bronchus.

Next, if we outline the upper lung border as we have done in percussion, (see figures 16 and 17) and place the bell of the stethoscope above this line posteriorly where there is no lung structure, beginning, as is proper, over the left side, we note distinct bronchial or tubular breathing, and having obtained a clear idea of bronchial breathing in the individual we are examining, we place the stethoscope a few cm lower, approaching the line and again carefully listening. We now note as we reach the lung border that the murmur begins to change, so that when we have

passed this line the murmur becomes distinctly vesicular, being entirely devoid of its bronchial quality. If now we compare these findings, that is the left side with the opposite, the right, we note that there is no such distinct and abrupt change when we pass the lung border on this, the right side, but that a distinctly bronchial quality is still heard with the vesicular and as we reach a point about 2 to 3 cm below the lung border the murmur again becomes distinctly vesicular; the murmur heard distinctly between these two points is the broncho-vesicular. This is normal in the right upper lung but never over the left if strictly normal. The same applies anteriorly below the clavicles, and in the mid-clavicular line of the left and right side. Other areas over which a more or less intensely bronchial quality may be added to the vesicular or a vesicular to the bronchial, depending upon which may predominate, are anteriorly over the body of the manubrium and on either side along the sternal end down to the 3rd rib and occasionally a few cm to either side, posteriorly in the interscapular area as far as the 3rd and 4th dorsal spine, and outward, perhaps a little beyond the paravertebral line and upwards to the upper margin of the lung. With the exception of this area posteriorly, and the area described anteriorly, and that portion of the lung extending above the clavicles over which broncho-vesicular or mainly bronchial is heard, over all other portions of the chest wall the vesicular murmur is in evidence, and any change from this indicates some pathological condition; any murmur heard over an area over which it is not heard in the normal indicates that something has taken place within the lung structure. As the normal lung is a poor conductor of sound, any infiltration will make out of this poor conducting media, the lungs, a good conductor, transmitting sounds more readily to the surface.

Abnormal auscultatory murmurs in pulmonary tuberculosis. The abnormal chest sounds heard in pulmonary tuberculosis are symptoms of infiltration. In the very beginning of pulmonary involvement the vesicular breath sounds begin to change. At first they may be accompanied by a decided weakening; this is soon followed by a distinct harshness and prolongation of the inspiratory murmur, the note acquiring a slightly higher pitch, the expiratory being still unchanged. However, a gradual and a greater change is soon noticeable in the expiratory sound which becomes still higher and more prolonged than the inspiratory,

the whole displacing entirely the soft inspiratory and still softer and much shorter expiratory of the normal vesicular. This is due, as has already been stated, to the changes in the lumen of the bronchiols, alveolar ducts and in the alveolar spaces, the infiltrate materially interfering with the free access of air, weakening first the vesicular murmur until it becomes gradually entirely lost.[1]

In the examination of the chest abnormal auscultatory sounds cannot always be diagnostically utilized, for in many women and quite a few men who are physically weak, abnormal sounds over the chest are often heard, for even the normal breath sounds are not always the same or heard with equal intensity over both sides in the same individual, even in perfect health. Attention must here again be called to the fact that the expiratory sound in the normal is higher over the right upper lobe both anterior and posterior especially on deep and full breathing. With deep and full inspiration over the upper left lobe posterior a slight prolongation of the expiratory sound also may be observed, which is not found on slightly superficial or moderate breathing. This expiratory phenomenon can only be utilized as a clinical sign when over the left apex posteriorly; over that portion over which the note was found high on percussion, the respiratory murmur is prolonged and harsh during very slight or moderate breathing or if it is heard only over a small and circumscribed area, as over healed lesions. In fibroid conditions the breath sounds also are intensified and prolonged and bronchial breathing frequently heard in the interscapular areas, over the upper sternum, about the trachea and above the clavicles has already been mentioned.

Auscultatory Catarrhal Signs. Dry and Moist Râles. Adventitious Sounds. Abnormal Chest Sounds. Catarrhal signs, usually crackling, heard over the chest are always foreign sounds and are produced by the respired air coming in contact with either abundant secretion or little moisture in either the bronchial tubes and their divisions or in the bronchiols, the terminal bronchi and the alveolar spaces.

In the very beginning of the disease numerous fine crackling

[1]In the very beginning, the inspiratory sounds become weakened and impure, but with very little if any effect on the expiratory. As the process continues, the expiratory sound also begins to change, becoming gradually more and more prolonged, more harsh and devoid of all soft quality; the pitch rises when eventually the process is complete; the vesicular sound has been completely displaced by the bronchial which is now higher in note than the inspiratory and as long or longer in duration. The student should remember at this point that the expiratory sound is the key to the diagnosis, not only in pulmonary tuberculosis but in all the other forms of pulmonary disease of whatever origin, whatever cause or nature.

sounds are heard which at the height of inspiration bespeak their catarrhal nature, especially if these sounds are heard over small circumscribed areas. Pulmonary tuberculosis is primarily a localized bronchitis, but not in the ordinary sense, being confined to the smallest tubes; more correctly is it a bronchiolitis, a bronchitis of the capillary bronchi. These numerous sounds, at first crepitating and fine crackling, usually heard at the height

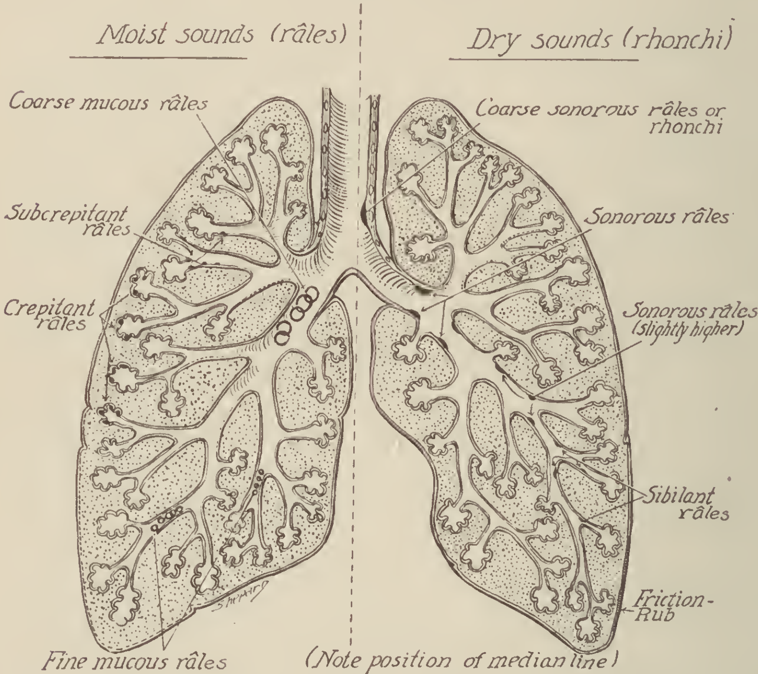


Fig. 21. Adventitious Sounds or Murmurs. (Râles, Rhonchi, friction rubs, etc.) A schematic representation of the various sounds heard over the chest cavity (abnormal). The nature and quality of these sounds indicate the particular location in the lungs in which those sounds are produced.

of inspiration, are known as crepitant râles. As the process extends to other tissues surrounding the initial involvement, invading contiguous tissue, the sounds begin to lose their fine crackling quality and at the same time lessen in the number of crackles. Fewer crackling sounds, known as subcrepitant râles, both inspiratory and expiratory, are now heard (in both phases of breathing), becoming larger, moist, and more of a mucous variety, and as the catarrhal process extends a cough, resulting from the heightened bronchial tube irritation, accompanies these sounds, and dry and moist râles are now clearly aud-

ible and sonorous and sibilant, mucous, subcrepitant and crepitant sound rapidly follow in ever changing order.

An explanation may not be out of place at this point. Sounds produced in the bronchial tubes of various calibre are often referred to by writers on physical diagnosis as musical râles. Admitting that the sounds produced in these tubes possess a musical quality, the time worn terms, sibilant and sonorous, although very old, and perhaps a little obsolete, are still better and more descriptive. If I am told by a student that over a certain area over the chest wall sibilant or sonorous râles (197) are heard, I know in what portion of the bronchial tubes and also in what size tubes these sounds have been produced, if in tubes of smaller or of larger calibre, but if I have been told that musical sounds were heard I must inquire about the quality of these sounds, if they were very high in note or very low, in order to orient myself.

Coarse râles having a metallic or coarse, crackling or creaking sound are usually evidence of small cavities, especially if accompanied by an expiratory prolongation and of low, hollow note; with large cavities the note becomes more amphoric or like the sound produced by blowing over the mouth of a bottle. These crackling or creaking sounds are heard usually in both the phases of inspiration and of expiration, but they are usually louder and more numerous with inspiration, more crackling, fewer and long drawn out with expiration. All fine crepitating as well as crackling sounds bear evidence of the catarrhal process in the small air spaces, the alveoli, the bronchiols and terminal bronchi whilst coarse and piping sounds produced in the larger tubes bear evidence of inflammatory secretions in these tubes.

Tuberculosis in the beginning is an alveolitis, and a bronchiolitis *per se,* and sibilant and sonorous râles if present are only evidence of an accompanying bronchitis or tracheitis. With the formation of cavities, these sounds change to creaking, short, sharp, crackling sounds, circumscribed, with the expiratory sound much prolonged, but not now as in early consolidation high pitched but low pitched and amphoric, that is, a bronchial, high pitched, expiratory sound speaks for consolidation but an amphoric, low pitched, expiratory for cavities.

In the opinion of some authors, a systolic murmur, which if heard over the subclavian artery on one side, especially if heard with expiration only, or if it becomes more intense in this phase is a sign which bespeaks of early pulmonary tuberculosis. The cause of this murmur is supposed to be due to adhesions of the apical pleura. A similar murmur, often in anemia, is heard in the supraclavicular fossa, but the sound is more intense with inspiration or it may be heard at the height of inspiration only.

Normal Vocal Sounds. Voice Resonance or Vocal Resonance. If we listen with the stethoscope placed over any portion of the chest wall over lung tissue both anteriorly and posteriorly, while the patient speaks or counts aloud, a peculiar vibratory sound is conveyed to the ear; this is known as the normal vocal or voice resonance. If next we place the bell of the stethoscope over portions of the chest behind which there is no lung tissue, but near which the bronchial tubes lie, a very harsh but muffled sound is carried to the ear; this is known as bronchophony or the bronchophonic voice. If now we ask the patient to whisper one, two, three while we are listening over the bronchial tubes, a distinct but less harsh sound is heard which is known as the whispering voice or whispering bronchophony.

These three sounds, normal vocal resonance, bronchophony or loud voice and whispering voice or whispering bronchophony in health, are heard distinctly over sharply defined areas, but if heard outside of these areas they usually indicate a condition of pathological lung change. As the normal vocal resonance is heard best over that area of the lung over which the vesicular breath sound is clearly heard, and bronchophony and whispered bronchophony over that part of the chest wall over which bronchial or tubular breathing is best heard, we can again make use of the outline of the upper and posterior lung border on the chest wall, as we did when considering the subject of percussion. (See figures 16 and 17.)

As has already been pointed out, on the posterior upper chest, the right and left lung, borders meet on a level with the second dorsal spine, extending from this point upwards and outwards on a level with the first and running from the second dorsal downwards and parallel with the spine to the 10th rib, in the axillary area to the 8th, and on the anterior chest to the 6th, and running thence upwards, the right from the 6th, and the left at the 4th, following behind the sternum, where at the junction with the manubrium, they again separate, running upwards and outwards and extending from an inch and a half to two inches above the clavicles. Over all this area, over which the lung structure lies superficially with the exception of that portion of the lung which lies above the clavicles, the normal vocal resonant sound is more or less distinctly heard, modified somewhat by the thickness of the chest wall, difference in muscular development, deposit of fat, bony framework, pitch of the voice, etc. This

resonant murmur is a muffled, humming, more or less loud sound, but over the trachea, bronchi and about the neck both the loud and whispering bronchial sounds are heard in the normal. If we draw a line along the posterior upper border of the lung from the second dorsal spine where both lungs come in close apposition, upwards and outwards on a level with the upper border of the first dorsal spine, we will outline, as has already been stated (on page 139) the upper margin of the lung. We draw a similar line over both the right and left side. If now we place the bell of the stethoscope anywhere above this line along the neck and ask the patient to whisper one, two, three or use the words, 99, 66, 33, the whispering voice or whispering bronchophony will be distinctly heard. This sound will continue over every square inch of surface above this line, but a decided change is observed when we pass this line. On the left side usually as we pass this line the change is abrupt, no whispering voice will be audible below this line but over the right side the sound is carried, though with less and less intensity, for about 2 or 3 cm below this line, when it also can no longer be heard.[2]

Abnormal Vocal Sounds. Whispering Bronchophony in Pulmonary Tuberculosis. The whispering voice sound in consolidation of the lungs is one of the most distinct and definite of all the physical signs; in fact, I consider it the most distinct single sign, and throughout the whole tuberculous process with the very beginning of the infiltration, with the earliest incipiency, the whispering bronchophony manifests itself, even long after the disease has been arrested or even if healed, the whispering voice will still be distinctly heard. The whispering voice is the very first sign to appear in pulmonary tuberculosis and the last to disappear. Whispering bronchophony heard anywhere over an area where normal lung structure is usually found indicates infiltration. To this there may be some exceptions, but careful examination will avoid errors. In beginning pleurisy, with effusion for instance, or even if the pleurisy has lasted for some time and the pleura is still thin, sounds are often conveyed which simulate consolidation. This will be described more in detail in the discussion of the subject of "Tuberculosis and Pleurisy."

In children the whispering voice is usually heard well between

[2]Caution. The student must learn to differentiate between the whispering sound heard through the stethoscope and that extraneous sound of the voice heard when the bell of the stethoscope is placed over any portion of the chest and the individual is asked to whisper. Practice alone will teach how to obviate such errors.

the scapulae, more particularly when the mediastinal glands are enlarged. This also will be referred to more in detail when the subject of gland tuberculosis is considered. On the anterior chest along the sternum occasionally to the right over the second and third interspaces, parasternal line and above the clavicles the whispering voice is also heard as normal.

Normal vocal resonance, as has previously been stated, is heard over healthy lung tissue as a dull, humming, low pitched sound; in healthy individuals the sound varies greatly in pitch and in intensity, is more feeble or diminished in women and children than in men and like all sounds produced depends greatly upon whether the voice is high or sibilant or screeching, or low, sonorous, a basso profunda. If the stethoscope is placed above the line so often referred to while on the subject of percussion and auscultation, say along the neck, about the larynx, trachea, thyroid, etc., and the person is asked to speak or count out loud, a most intense, a more or less painful sound is distinctly heard. This is normal bronchophony; the words are still confused, not perfectly clear. Over slightly consolidated and infiltrated lung areas, similar sounds are usually heard, still muffled, but with complete consolidation or with the formation of cavities, these sounds may again change and the voice is then distinctly and clearly transmitted through the stethoscope to the ear; this is described as pectoriloquy or clear voice conduction. Generally in consolidation, pectoriloquy is heard more sharply and over larger areas, high in pitch, more intense and nearer to the ear, while pectoriloquy heard over cavities is more circumscribed, over smaller areas, lower in pitch, less intense, more distant from the ear. If such conditions are present then both forms of pectoriloquy can usually be demontrated about these areas, because the area surrounding cavities is usually densely consolidated and a good conductor of sound. Here also the whispered voice or whispering pectoriloquy is most definite and distinct as in simple voice conduction.[3]

How and When to Auscultate. It is most important that aus-

[3]It is often very easy to demonstrate on many healthy and normal individuals normal vocal resonance, bronchophony and pectoriloquy. Place the bell of the stethoscope on any part of the posterior chest, say, near or below the angle of the scapula and listen while the patient speaks; this low muffled sound heard is the normal vocal resonant. Next place the bell anywhere about the neck, say, above the line so often mentioned and the loud sound heard is the bronchphonic voice or simply bronchophony, the voice less muffled more intense than the former but still not quite clear; now carry the bell of the stethoscope to the occiput or even on top of the head and the sound heard there is the clear and distinct voice, perhaps somewhat distant and faint; that is pectoriloquy. (See Figure 17, page 139.)

cultation be practiced at a time when we may anticipate the best results, especially in beginning cases of pulmonary disease when the adventitious sounds can be definitely located. This time is usually best in the early morning when the patient is endeavoring to clear the bronchial tubes from the secretions which have accumulated during the night. The patient should then be examined first under ordinary breathing, then under forced or deep breathing, paying close attention to the first sounds or râles which may be heard at the height of inspiration. Next, carefully auscultate the cough. No examination can be said to be well done if this has been omitted. The patient is asked to take a deep breath, a full inspiration, and at once to exhale or breathe out, coughing just before he again takes another breath, then immediately to follow this by a deep inspiration, when crackling sounds may be heard with the height of the inspiratory phase. It is really not auscultating the cough, but the inspiratory phase immediately following. Coughing quickly at the end of expirating expells a great amount of residual air, collapsing the small bronchial tubes, the bronchiols, the terminal bronchi and aveoli which in the next, the inspiratory phase, are forced asunder by the entering air and due to the sticky mucous contained within their lumen, produce crackling sounds of various sizes. Auscultation of the mouth may furnish in some cases very reliable data. By placing the bell of the stethoscope before the mouth of the patient while breathing in slowly and quietly, but deeply, crackling sounds are often distinctly heard which usually point to a deep seated or central lesion. It is advisable that women be examined during the absence of the menstrual period.

Errors in Auscultatory Sounds. Not all sounds which are heard with the stethoscope when listening over the thoracic wall can be construed as having their origin in a normal or pathological chest. The student can learn only from constant observation to know the difference between these various sounds produced. Many such sounds heard over a part of the chest wall are extraneous and are usually produced at a point more or less remote from the point at which they are heard. Some such sounds, for instance, may be produced over the opposite side, but conveyed to the side over which we are listening; again, sounds produced in the upper air passages during the act of breathing, in the nose, the mouth, the pharynx or the larynx, may also be distinctly heard over the chest and may be construed

as being produced just below the point over which we are listening. Still other sounds leading to error are such as may be produced by the bell of the stethoscope pressing against the skin, especially in such cases in which the skin is dry or rough, by the rubbing of the hair or by the movements of the muscles. These latter the so-called muscle sounds are usually lost if the muscle producing the sound is made to relax, as, for instance, if the muscle sound is heard posteriorly in the upper portion of the chest; if we change the position of the arm either up or down or sideways, the muscle sound will be lost. These muscle sounds are very frequently mistaken for pleuritic friction rubs, but their inconstant nature and very superfiicial sound are sufficient for identification. Further, muscle sounds are usually present during inspiration and decreased or entirely absent during expiration. The muscles of the back and shoulders, by contraction and movement of the scapulae may all simulate muscle sounds; these also are often lost by a changed position of the arm on the side where these sounds are produced.

The sounds of tracheal breathing are heard chiefly along the spine and with equal intensity over both sides. Harsh breath sounds are also often heard over normal lungs if breathing be continued while the mouth is closed, if the chink at the larynx is much contracted or abnormally small; if free breathing is interfered with by pushing the head too far forward or downward during the examination, or by the noise made while breathing through the nose, usually at the nasal opening anteriorly. It must also be noted that in contracted bronchiectatic conditions which are usually the result of a healed tuberculous process, crackling râles simulating a recent tuberculous process may be heard almost indefinitely; at the base of the lungs fine, crackling inspiratory râles are frequently heard, due to pulmonary atelectasis, which after repeated and deep inspiration usually wholly disappear. These atelectatic râles may not be heard only at the base but along any border of the lung if the free excursion is interfered with from any cause.

The student is here again to be reminded that in the diagnosis of pulmonary tuberculosis, the evanescent or changing râles are of no import, that permanent crackling sounds only must and can be utilized because evanescent sounds or râles as we hear them in atelectasis, in which by insufficiently deep breathing the lower margin of the lung structure has not been separate for

some time, usually clear up upon a few full and deep inspirations. This also applies to the crackling sounds occasionally heard at the apices of the lungs in acute and in chronic catarrh, in emphysema, etc., which are not always due to tuberculosis, but to simple catarrhal conditions; here again the evanescent nature of the râles is of prime importance. In collapse induration of the lungs, the result of faulty breathing in the upper air passages, the tonsils, adenoids, turbinates, etc., crepitating and crackling sounds are also heard over the collapsed area. These also are of a more or less evanescent nature, the patient presenting a very different clinical picture from the tuberculous.[4]

In examining individuals for tuberculosis we are constantly making use of the physical sensitiveness of feeling, seeing and hearing, but the sense of smelling and tasting are not applied. If not the sense of taste, has not the sense of smell a place in physical diagnosis? Many persons suffering from active tuberculosis have about their person an odor which is quite characteristic. In the exudative form of the disease with high fever and rapid pulse this odor is particularly noticeable. To my sense of smell this odor is akin to tuberculin which for some time has been exposed to atmospheric air (rancid). This odor is so distinct and definite that if recognized at a physical examination I state that it is pathognomonic of active pulmonary tuberculosis. I am so positive of this that in preparing a patient for examination (stripping to the waist) if I recognize this odor I make in my mind a positive diagnosis of active pulmonary tuberculosis without physical examination. This has often been confirmed by the subsequent course of the disease.

The Mechanism of Breathing[5]

The mechanism of breathing and the respiratory sounds produced during the act of breathing have often lead to misinterpretations. If we observe a normal, healthy adult while asleep or resting, we will note that breathing begins with a moderately full inspiratory phase; this is immediately followed by one of expiration, then a pause, a rest, then the beginning of another inspiratory phase, etc. This inspiratory phase, followed by an expiratory one, and then by a period of rest is known as a cycle of breathing, and the whole is spoken of as the Mechanism of Breathing. We will also note that the inspiratory phase is a short one, the expiratory a trifle longer, and that the period of

[4]The pitch, duration and intensity of the sound waves may best be exemplified by the following simile. A large steamer is approaching the harbor. It makes its presence known by blowing a steam whistle which gives a sound of deep, low, sonorous tone and which is heard at a great distance. A little tug in the harbor answers the signal by a whistling but sibilant sound, short, quick, high. Its waves are carried but a short distance.

[5]To make clear to the student the factors concerned in the physical examination of the chest by means of sight and hearing.

rest is a little longer than both inspiration and expiration combined. These cycles of breathing are rhythmical and are repeated in the normal subject about sixteen to twenty times a minute, with about four heart beats to each cycle of breathing. This is what we observe on inspection, and it should not be confused with what we hear on auscultation.

On auscultation over the normal chest, we hear different and distinct breathing sounds during each cycle, over different parts of the chest. Placing the bell of a stethoscope over the upper sternum or spine, we hear a harsh high-pitched inspiratory murmur during the whole of the inspiratory phase, and an equally harsh murmur, not so intense, higher-pitched, slightly longer during the whole of the expiratory act after which nothing is heard during the period of rest until, with the beginning of the next cycle, similar sounds are again heard; and so on with each following cycle. This is known as bronchial breathing.

If in another cycle we place the stethoscope below the clavicle on the left side, about the second rib in the mid-clavicular line or, posteriorly, near the angle of the scapula either right or left, we will hear with beginning breathing, an inspiratory sound much softer and lower in pitch than the one we heard over the upper sternum or spine. This sound is also heard during the whole of the inspiratory act; with expiration a still softer and lower sound is heard. This expiratory sound is not heard during the whole of the expiratory act as we found when listening over the upper sternum. It is heard only during one-fourth or one-third of the act, when all sounds cease, though the act of expiration is still going on; after this follows the period of rest. Now, if we compare the area over the upper sternum with the one below the left clavicle, and study the sounds carefully, we will obtain a good picture of what is meant by bronchial breathing and by vesicular breathing.

After we have a clear conception in our mind concerning these two phases of breathing we next place the stethoscope below the clavicle on the right side towards the parasternal line and pay close attention to the sounds we hear there. We will notice that during the inspiratory phase we hear a sound that is not quite so harsh nor as high and intense as the bronchial, nor quite so soft and low as the vesicular. This is the broncho-vesicular inspiratory sound. The expiratory sound heard immediately following is also not so high as the expiratory bronchial, nor so soft

or as short as the expiratory vesicular and is known as the broncho-vesicular expiratory sound. The broncho-vesicular murmurs are generally of two types; if the bronchial sounds predominate they are broncho-vesicular; if the vesicular are most prominent, they are vesiculo-bronchial.

The student should remember that the cycle of breathing is what he sees on inspection, but the breath sound produced during each cycle of breathing is what he hears through the stethoscope. This is all graphically outlined in the following diagram in which the two circles represent a cycle of breathing, and the three unequal semicircles within these circles represent bronchial, broncho-vesicular and vesicular breathing.

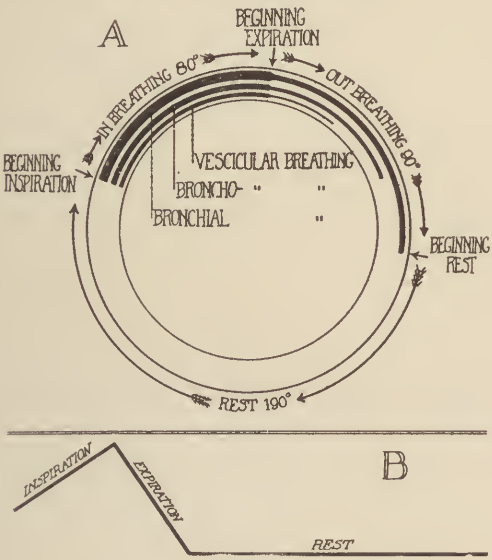


Fig. 22

A Cycle of Breathing. Normal Breathing Rhythm

(A) The two circles represent a complete cycle of breathing. This is what we see when we observe breathing in a healthy adult.

The three semicircles within represent the sounds which we hear when the bell of a stethoscope is placed over the chest of the normal adult during respiration.

The longest semicircle shows bronchial breathing, heard during the entire respiratory act. Inbreathing is harsh, high pitched and prolonged, outbreathing less harsh, higher pitched and slightly longer. The shortest semicircle represents vesicular breathing. Here also the sound is heard during the whole of the inspiratory act, but it is very soft as compared with the bronchial, lower in pitch and of equal duration, the expiratory sound, however, differs greatly from the expiratory bronchial. Here the sound is found very soft, very low in pitch and of very short duration. As compared with the bronchial it is only about one-fourth as long and is heard during a very short period after beginning expiration. Broncho-vesicular, also known as atypical intermediate or indeterminate breathing is midway between, is represented by the middle semicircle and may give the quality of one or the other depending upon which predominates the vesicular or the bronchial.

(B) A cycle of breathing as is usually expressed by two vertical lines and a horizontal line. [Inspiration 5, Expiration 6, Rest (apnea) 13.]

CHAPTER 17

ROENTGENOLOGY

The Relation of X-Ray to the Diagnosis of Pulmonary Tuberculosis

Historical Data. The value of X-ray in the examination of chest conditions was recognized very soon after Prof. Wm. K. Roentgen discovered this form of radiation in December, 1895. Due to the long exposure necessary, at that time, to obtain plates, only gross changes could be demonstrated. Interpretation of these plates was often difficult because the shadows were blurred by respiratory movements.

In 1905, Rieder and Rosenthal (189) exhibited X-ray plates taken while the patient held his breath. With this began a new era in the X-ray diagnosis of chest conditions. Fine details were shown on plates and slight changes recognized. The use of the Roentgen ray in chest examinations spread quickly, and instead of its occasional employment, it is now depended upon in a large percentage of cases.

General Consideration. Knowledge of the X-ray interpretation of pulmonary conditions has been acquired by carefully checking the fluorescent screen and plate findings with those of the clinical, physical, and laboratory examinations, and by autopsy study, both gross and microscopic. Withal it is still fallacious to believe that the X-ray furnishes the method *par excellence* for making the diagnosis of all chest conditions. It is sometimes the means of arousing suspicion and stimulating further clinical study, and it may be the only means for discovering intrathoracic pathology, as, for example, an early central pneumonia, an incipient pneumothorax, or a foreign body.

A complete X-ray examination of the thorax is best done by fluoroscopy and plates, preferably stereoscopic. Each examination has its own specific value, and each supplements the other.

Fluoroscopy permits, by changing the position of the patient, a view of the thoracic viscera and their movements from different angles. Especially noticeable are the movements of the

heart and diaphragm. Pathological conditions which make relatively gross changes may also be noted and an idea of their location obtained. Certain changes are better shown by fluoroscopy than by plates, such as hydro-pneumothorax or irregularities of the diaphragm and its movements.

Plates bring out much finer detail and furnish a permanent record so important for comparison in repeated examinations. A plate shows only the condition present at the instant of the exposure, and additional plates must be taken to show changes in position or motion. Stereoscopic plates add the factor of perspective and permit fairly definite localization of thoracic lesions.

An X-ray plate is a record of the differences in density of the objects rayed. The denser the object, the more it obstructs the ray, and the more definite, or heavier, will be the shadow cast on the plate. If, during plate exposure, a direct ray must pass a dense object overlying one less dense, the latter is obscured more or less completely by the former, as, for example, the heart overshadows certain of the bronchial markings. If two practically equal densities have certain portions in line with a direct ray, their combined density in that particular area will cast a considerably heavier shadow, as, for example, the area of intersecting rib shadows.

(A) The Normal Chest Plate. Markings, Shadows and Findings

It is very important to know the normal shadows on a plate or screen in order to recognize a deviation therefrom. A systematic routine is needed in X-ray interpretation to avoid overlooking apparently minor, but often important, details. The following outline is suggested:

1.—The diagnostic quality of plate or screen.
2.—Contour and subcutaneous structures.
3.—Bony structures.
4.—The diaphragm.
5.—Mediastinum, including cardio-vascular apparatus.
6.—The hilus shadows.
7.—The lung markings.

Plates of adults are here considered and will be described with interpolation of screen findings.

Normal Findings

1. **The diagnostic quality of plate or screen.** The diagnostic quality of a plate depends upon the technic used. Plates should

be taken while the patient holds the breath to avoid blurring by respiratory movements. Forty to sixty milliamperes, 4½ to 5 inch spark gap, 28 to 30 inch distance from target to plate, and 1 to 2 or 3 seconds' time will give a good plate. The higher settings, longer time, and greater distance are used with thick patients. Some feel that as nearly an instantaneous exposure as possible is preferable in all cases which require even higher settings than close given time.

Routine plates are better taken in the postero-anterior position, preferably with the patient sitting so that the diaphragm

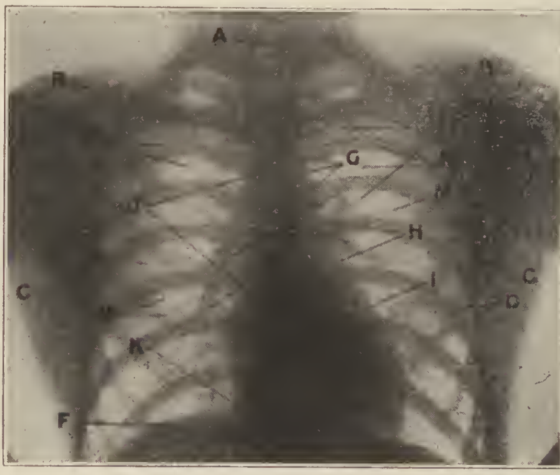


Fig. 23. The normal chest plate. Explanatory note: (a) Trachea; (b) Clavicles; (c) Scapulae; (d) Rib; (e) Calcification of costo-chondral junction; (f) Diaphragm; (g) Aortic Arch; (h) Pulmonary Artery and Appendix of the left auricle; (i) Left Ventricle; (j) Ascending Aorta; (k) Left Auricle; (l) Hilum; (m) Lung Markings.

and heart descend. The arms should be elevated to remove the scapulae as far as possible from the lung fields. The tube is centered over the spine at the level of the lower borders of the scapulae. For stereoscopic plates, the tube is shifted six cm upward for the second exposure. Parts nearer the plate are more clearly shown, hence the postero-anterior position gives a better view of the interior of the thorax, especially the apices. The posterior parts of the ribs, the scapulae, and the upper dorsal and neck muscles are then farther from the plate and their shadows therefore less distinct. The anterior intercostal spaces are wider than the posterior, and the cartilaginous portions of the ribs (unless calcified) do not cast a shadow, hence a less ob-

structed view of the intrathoracic shadows is obtained on postero-anterior plates.

A successful screen examination depends upon the proper amount of X-ray penetration for the thickness of the patient rayed. Two to four milliamperes and 5 to 6 inch spark gap are usually sufficient. Thick patients require more penetration, even up to 5 milliamperes and 6 inch spark gap. Very thick patients are poor subjects for fluoroscopy. Care must be taken to avoid burns. A one to two millimeter aluminum filter on the fluoroscopic table is a wise precaution.

The examiner's eyes must be thoroughly accommodated to the dark, as otherwise many details escape notice. The average person should remain in the dark five to ten minutes, or until, for example, small dots on a "radio" watch are seen to illuminate brilliantly.

2. **Contour and subcutaneous structures.** The contour of the two sides of the thorax is equal and symmetrical. The thickness of the subcutaneous structures can be determined only at the lateral margins of the thoracic shadow, and is an indicator of the state of nutrition of the individual rayed. On plates of females the breasts cast rounded sharply defined shadows (See Fig. 24-a). If large they may interfere with a clear view of the diaphragm and lower parts of the lungs. During fluoroscopy, they may be pushed aside.

3.—**The bony structures.** Normally the spine appears straight in postero-anterior and antero-posterior views. The vertebral bodies are seen fairly distinctly in the region of the trachea, less distinctly or not at all in the portion overshadowed by the sternum and cardio-vascular apparatus.

The trachea (Fig. 23-a) is represented by an area of decreased density, 1 to 2 cm wide with straight margins, seen in the midline of the cervical and upper dorsal spine. Under favorable conditions, the bifurcation of the trachea and even the large bronchi may be made out. The upper portion of the trachea is deviated to the right or left of the spine if the patient's head is turned to the corresponding side during the exposure.

The clavicles (Fig. 23-b) extend upward and outward from the sternum to the shoulders. The right is nearly always a trifle higher than the left. The inferior angles and lateral borders of the scapulae (Fig. 23-c) are seen outside the thoracic margin if the patient's arm were elevated during the exposure. The verte-

bral borders and especially the spines of the scapulae are not so well seen in the postero-anterior position as in the reverse. Remembering these findings makes it easy to determine the position of the plate during exposure. The ribs (Fig. 23-d) are distinctly outlined and symmetrically placed. The width of the interspaces gradually increases from above downward, and is wider anteriorly than posteriorly. Due to their curve around the thoracic wall, the rib shadows on the flat plate intersect, giving a latticed effect. On stereoscopic view the ribs assume their normal perspective. Trabeculae are usually seen in the parts nearest the plate. Only on very favorable plates do parts farther from the plate show trabeculae.

Calcification, if present, makes either a dense line at the costochondral junctions (Fig. 23-e), or an irregular shadow in the cartilage. Calcification is often present in adults, usually increasing with the age of the patient. The cartilage of the first rib may be the only one involved, or, if all are involved, the first is usually more densely calcified than the others.

Plates taken with the patient's left side against the plate (exposure through the right oblique) give a lateral view of the spine and bring out the thoracic curve, and each vertebral body is then quite distinct. The prevertebral space, or posterior mediastinum, through which the oesophagus passes, is clear. In plates made with the patient's right side against the plate (that is through the left oblique) the heart shadow covers the spine and obscures the prevertebral space in the lower thoracic portion.

On screen examination, the respiratory mobility of the bony structures can be determined. Bone detail, however, is less distinct than on plates. The clavicles and anterior portions of the ribs move through a range of about two centimeters on deep respiration. The posterior portions of the ribs move but little. Elevation of the arms of the patient gives a clearer view of the lungs by partly removing the scapulae from the field.

4.—**The Diaphragm.** In the postero-anterior view, the diaphragm (Fig. 23-f), as seen in each lateral half of the thorax, is dome-shaped, smooth, and higher on the right side—in this plate, 1.5 cm. It forms an acute angle on each side with the lateral thoracic wall. The angle formed with the heart shadow on either side is less acute, in some chests even slightly obtuse.

Movements of the diaphragm are observed during screen examination. On quiet respiration its excursion is 1 to 2 cm. In

patients with the thoracic type of respiration, the movements of the diaphragm are but slightly increased on deep inspiration. In those having the abdominal type of breathing, the diaphragm descends uniformly 5 to 7 cm on deep inspiration. The angles with the lateral walls remain acute, the angles with the heart become more obtuse.

5.—**Mediastinum, including cardio-vascular apparatus.** The chief mediastinal shadows are those of the heart and great vessels. Normally, these vary in size and shape within rather wide limits depending upon the height and weight of the individual. The two extremes are the long, narrow, "drop" heart, and the transverse or "squat" type. Variations between these are within the range of normal. The more frequent type, is here described.

Just below the sterno-clavicular junctions, and on a level with the fourth and fifth thoracic vetebrae, lies the aortic arch (Fig. 23-g). Its shadow is rounded and definitely outlined and it covers and extends a little beyond the spine shadow on each side, forming a knob-like projection on the left. The width of the aortic arch shadow is approximately one and one-half times that of the spine.

On the left, just below the aortic knob, is a second, smaller shadow (Fig. 23-h) with a slightly more elongated curved outline, (or rarely, normally, two smaller successive curves), caused by the pulmonary artery and appendix of the left auricle. This shadow merges with that of the left ventricle (Fig. 22-i) to form the major portion of the heart shadow. The left ventricle outline is curved and extends downward and outward till the apex lies at the apparent intersection of the fifth rib anteriorly with the ninth rib posteriorly—in this case 9.5 cm to the left of the midline of the spine. From this point, it curves again toward the spine, and meets the diaphragm shadow near the midpart of its dome. The descending portion of the aorta passes downward from the arch to the diaphragm. The left margin of its shadow is sometimes seen through the heart area—the right margin is hidden by the spinal density.

On the right, the nearly straight edged shadow of the ascending portion of the aorta (Fig. 23-j) lies close to the right margin of the spine (here within 1-1.5 cm). At the level of the eighth dorsal vertebra, the right auricle outline (Fig. 23-k) curves beyond the margin of the ascending aorta and downward to the diaphragm. It extends outward to the right of the spinal margin

approximately 4 cm in the widest portion. The right ventricle outline is not seen. The vena cava is sometimes seen as a straight shadow crossing the right cardio-diaphragmatic angle close to the spine.

During the fluoroscopy the pulsation of the heart is seen as a rhythmic movement of its shadow. The aortic pulsation is also seen. It has less excursion than that of the heart. During full expiration, the heart shadow is pushed upward and is widened, thus assuming a more transverse position due to the elevation of the diaphragm. On deep inspiration, the heart shadow becomes longer, and more perpendicular, and narrower, due to the depression of the diaphragm and consequent change in the position of the heart. At the same time, a narrow, clear space appears between the left side of the diaphragm and the lower border of the heart.

Viewing the patient from in front through the right oblique, the cardio-vascular shadow is separated from that of the spine and is seen in almost directly lateral view. The aortic arch then appears longer and narrower, about one-half to two-thirds the former size. The right margin of the heart follows practically the same curve as the spine. The left heart border is distorted and extends outward well toward the lateral thoracic wall, usually to within 2 to 3 cm.

6.—**The Hilus Shadows.** The hilus shadows (Fig. 23-1) are masses of density lying in each half of the chest near the midline and at the level of the sixth and seventh thoracic vertebrae, or in other words, at the lung roots. They are composed of the conglomerate mass of shadows of the large bronchi, arteries, veins, and peribronchial lymph glands. The outer edges have a tendency to linear radiation, corresponding to the heavier bronchial trunks. Normally, hilus shadows vary considerably in size and density in different individuals. Both generally increase with the age of the patient. Very dense rounded shadows are also frequently present, and correspond to the calcified peribronchial glands found in nearly all persons at autopsy. Hence, on plates they have the same significance. In Fig. 23, one large and one small dense, rounded shadow is present on each side. The large one (1 cm diameter) on the right is made up of a cluster of smaller shadows.

On screen examination, hilus shadows are irregular masses of more or less heavy density about the lung roots. If the patient

is turned to the left, semilaterally, the right hilus density is usually lost in the spine shadow and the left hilus density is lost in the heart shadow. If, however, the hila are large and dense, or if the patient is turned farther to the left, their shadows may be seen in the region of the posterior mediastinum.

7.—**The Lung Markings.** Lung markings (Fig. 23-m) are quite distinctly seen on plates and consist of branching linear shadows caused by the bronchial tree and its neighboring structures.

Beginning at each hilum, the right and left bronchi divide into their main trunks, usually three on the right and two on the left side. The shadows of these with their spreading subdivisions extend outward in a triangular arrangement, apex to hilum and base toward periphery—the arrangement sometimes called a "fan."

The first trunk on either side divides into the vertebral and first, second, and third interspace branches. The shadow of the vertebral branch extends upward toward the apex. On the right it lies close to the spine, on the left it is a little farther out, due to the fact that the left bronchus divides after curving under the arch of the aorta. The shadows of the next three branches on either side lie in the plane of the first, second, and third intercostal spaces, and are named from these positions. The finer branches more or less intermingle or overlap, especially those of the second and third interspace branches. The middle trunk on the right supplies the middle lobe, while on either side the remaining trunk divides into the lower lobe branches, which as seen on plates, radiate downward and outward. All branches are best made out on stereoscopic plates, together with some additional (127) details not otherwise observable.

Normally the shadows of the various bronchial branches can be followed about two-thirds the distance toward the periphery beyond which the branches become so fine and so nearly the same density as the surrounding lung tissue that they do not cast a distinct shadow on the plate. Lung markings are practically equal in density from above downward and in corresponding positions in either lung, except that the lower lobe branches are large and heavy near the spine. They are harder to see on the left because they are overshadowed by the heart. The apices (within the circle of the first rib) are clear and show either no, or but faint, markings of the bronchi. It is especially to be noted

that no fan stands out as heavier than the one above or below. With advancing age of the individual rayed, all branches are more dense and extend nearer to the periphery, including the apices.

On screen examination the lung areas are practically clear from apex to base. The density of corresponding portions of the two sides is equal. If conditions are very favorable, some of the coarser lung markings are distinguished as linear strands radiating outward from the hila. Finer markings are lost on fluoroscopy. On deep inspiration, the lung areas "light-up" or appear less dense practically equally on the two sides.

(B)—**Variations from Normal.** Interpretation of pathological chest findings in tuberculosis. (167)

Variations from normal plate shadows consist in either increased or decreased density, with or without distortion of the outlines or organs. Variations producing an increased density may be due to pathological changes which have taken place, either within the lung parenchyma or along the bronchi.

The type of the variations giving an increase in density, and due to pathological conditions along the bronchial tree and its surrounding parenchymal structures, may or may not produce on plates shadows which follow closely the bronchial branches. The shadows which do follow the bronchial branches give a triangular or fan-shaped arrangement, with base toward the periphery and apex toward the hilum. If such shadows are in upper lobes, if their density is mottled and extends well toward the periphery, and if one or more heavy bronchial shadows connect with the hilum—which also shows increased density—a tuberculous infiltration of the lung is suggested (142).

When the linear markings of one or two upper bronchial branches are slightly heavier than those of the neighboring branches and when the peripheral portion shows a hazy and slightly stippled, or mottled appearance, an incipient or very early tuberculosis is to be considered. Such changes are frequently at the level of the first or second anterior interspace. In other cases, the increased linear markings extend into one or both apices, and may, or may not, end in small, rounded, denser shadows. In still other cases, the increased density of the linear markings is absent and only a haziness or clouding of one or both apices is found. If both apices are involved, the shadow in one is usually denser than in the other. Dunham (146) stated (103)

that the right apex is involved before the left, judging by its heavier density as seen on X-ray plates, in the proportion of 55% to 45%.

Associated with any of these variations, the corresponding part of the hilus shadow may be increased in density and often includes small, round, dense shadows due to tuberculous changes. In cases with clouding of the apices only, other reasons for the clouding must be considered, such as shadows caused by heavy neck muscles, thickened pleura, supra and infra-clavicular glands, struma, scoliosis, cervical ribs, movements, or an artifact. Clinical findings are, therefore, of great importance in the interpretation.

If the shadows in or near the apex are due to an active tuberculosis, corresponding physical findings are present, such as dulness, harsh or bronchial breathing, and râles, also general symptoms, such as cough, loss of weight, failing appetite, etc. The slight changes so far described are rarely, if ever, recognized on screen examination.

Fig. 24. Early Tubercu'osis. First Stage. (a) Breasts; (b) Heavy Lung Markings.

Fig. 24 illustrates a typical case of slight variation from normal. The right apex is hazy. No linear markings extend into it. The linear shadows of the bronchial branch in the right first interspace are heavier and extend farther toward the periphery than those of any other branch in either lung. Near the periphery, the markings are not well defined, but are hazy and somewhat

stippled. The bronchial markings in the left first interspace are nearly as heavy and extend nearly as far towards the periphery as those of the right, but here is neither haziness or stippling. A heavy linear marking is present on the right side (Figure 24-b), and extends downward from the hilum well toward the diaphragm. The hilus region on either side is moderately heavy and contains three or four dense rounded shadows in each. These lie near the apices of the triangular areas of increased density just described.

On physical examination, fine crepitant and subcrepitant râles were heard anteriorly and posteriorly over the area corresponding to the shadow in the right first interspace. On the left, over a small area in the third and fourth interspaces near the sternum, occasional subcrepitant râles were heard. A friction rub was present at the lower border of the left lung. (Left sided pleurisy was present six months previously. No corresponding pleural shadow is made out on the plate.) There was no cough or expectoration, temperature to 99.6°, pulse 70-80. Complement fixation test for tuberculosis + + +. Diagnosis, based on the combined findings —early tuberculosis.

Increased density of the bronchial markings is also found in other chronic inflammatory processes, such as bronchitis and asthma, or due to long continued inhalation of coal-dust or similar material. In plates of such cases, the linear markings are increased in density and extend farther toward the periphery than normal, but in distinction to tuberculosis, no one or two branches stand out as heavier than all others; instead, the markings are practically uniform throughout both lungs, though occasionally the markings of one lung are a little heavier than those of the other. Haziness of the peripheral portion of the bronchial branch shadows is absent, or, rarely, very slight. Stippling, when present, is uniformly distributed, and occurs in small, rounded, fairly dense spots, with little or no surrounding haziness.

Advance of the tuberculous process accentuates the previously described plate findings. Increased density is seen in several of the bronchial branches, usually of the upper lobes, with variation in the degree of density in the different triangular areas. The lung markings appear more broken and irregular, due to being overshadowed by larger flake-like spots. The general appearance of these shadows is that of a heavy snowstorm which grows lighter toward the lower lobes. Increase of the density and of the size of the flakes bespeaks the advancement of the process. In general, those bronchial branches first affected cast

the heaviest shadows. Rarely are the shadows in the different areas uniform. In fact, the irregularity speaks strongly for tuberculosis. (Areas in which the density is heavy need not necessarily be "active" in a clinical sense, but may be of long standing, or partly healed).

The corresponding areas of the hilum show heavier shadows somewhat in proportion to the increase in density elsewhere in the lungs. Usually there are one or several rounded, very dense shadows in the hilum, due to the affected lymph glands. It is not unusual to find a high position of the diaphragm on the affected side.

A tuberculous process, sufficiently advanced to give such plate findings as are here described, also gives definite clinical findings, such as productive cough with tubercle bacilli in the sputum, fever, rapid pulse, emaciation, night sweats, etc., also dulness, bronchial breathing, and râles. The greater dulness, tubular breathing, and coarser râles go with the greater density and stippling in larger, heavier flakes. The slight dulness, slight bronchial breathing, and fine, crepitant and subcrepitant râles are associated with the slight increase in density, slight haziness, and stippling in small, not very heavy spots. In general, the latter findings thus become evidence of a more recent or acute process, while the former are evidence of one older or more advanced.

On screen examination, the density of the lung field varies in the affected areas from heavier than normal to very dense. Often there is also a blotched or spotted appearance consisting of lighter and darker areas. These variations in density correspond to the amount of infiltration of the affected bronchial branches. Limited respiratory excursion and irregularity in outline of the diaphragm are also frequently seen.

Figures 25 and 26 are from plates of the same individual.

The patient's main complaints were difficulty in swallowing, associated with shortness of breath on exertion, persistent cough and expectoration, some loss in weight, and profuse night sweats. The difficulty in swallowing was due to cardiospasm with retention of as much as 375 cc of food and mucus in the oesophagus. Lung expansion was poor, and dulness was present over both upper lobes. Many râles were heard on both sides anteriorly and along both sides of the spine. Tubercle bacilli were found in the sputum.

A plate taken at this time (Fig. 25) shows a triangular, uneven density on the right side, in the region of the apex and first and second interspaces. The peripheral portion of this shadow is

denser and more hazy than elsewhere, and is densest in the second interspace. Extending downward from the apex to the diaphragm is a snowstorm of moderately large, flake-like spots which grow progressively lighter and smaller toward the base.

A similar triangular, uneven density is present on the left side, also involving the apex and first and second interspaces, but is not so heavy as on the right. Smaller, less dense, flake-like spots are present. These are scarcely noticeable below the level of the third rib anteriorly. About half way to the periphery in the left second interspace is a pear-shaped area of decreased density, (2x4cm) with the narrowed portion outward. This area is surrounded by a fairly dense, slightly irregular margin. These findings are characteristic of a cavity. On screen examination a heavy, mottled density was present, especially in the upper lobes.

Fig. 25. Advancing Tuberculosis. Cavity Formation. (A) Cavity.

After receiving treatment for the cardiospasm, the patient gained thirteen pounds in five weeks. In the plate taken at this time (Fig. 26) the general density of the triangular shadows in the upper lobes is heavier than in the previous plate (Fig. 25). The flake-like spots, giving the snowstorm appearance, are larger and denser, and are seen throughout the right side and extend a little farther down on the left. The left lower lobe is still practically clear. In the left second interspace, there is a heavy, nearly solid density, somewhat triangular in shape, with apex

outward, extending from the hilum about two-thirds the distance toward the periphery. (This is the area in which the decreased density—cavity—was seen on the previous plate.)

Since the snowstorm appearance in Fig. 26 involves a greater area of the lung fields than in Fig. 25, an advancement of the process is clearly indicated. The increase in density of the upper lobes is difficult to interpret from the plate alone. It may be due entirely to advancement of the tuberculous process, or may be due in part at least, to healing. Since eleven months later this patient had gained 33 pounds and was "feeling fine," it is reasonable to believe that a healing process had begun at the time Fig. 25 was taken. This brings out the difficulty of correctly inter-

Fig. 26. Active Tuberculosis. Far Advanced. Note area of cavitation. (A) Cavity Area.

preting the condition of the patient from plates alone, and emphasizes the importance of combining the clinical evidence with the plate findings.

(C) Differential Diagnosis.

In the differential diagnosis the pneumonias are chiefly to be considered.

The shadow of lobar pneumonia is a uniform "pneumonic" haze, veiling the lung markings more or less completely, (Fig. 27-a). The shadow is triangular in shape with apex outward, and usually with at least one straight, sharply defined margin corresponding to an interlobar septum. Part of a lobe, especially in the early stages of the disease, later an entire lobe, or more than

one lobe may be involved—lower lobes more frequently than upper. The shadow varies markedly on re-examination within a few days, due to the rapidly changing stages of the pneumonia.

The history and physical findings are usually characteristic for a pneumonia. In clinically atypical cases, the plates findings may speak definitely for pneumonia, especially an early central pneumonia, some time before other findings warrant such a diagnosis.

The shadows of an unresolved pneumonia, or one in which resolution is irregular or delayed, may very closely simulate those of tuberculosis, especially if involving an upper lobe. In such cases the differentiation must be by clinical means.

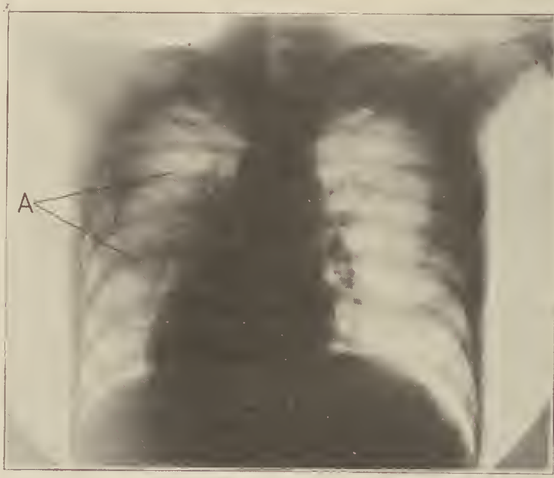


Fig. 27. Pneumonia. Note area of congestion. (A) Area of Congestion.

Shadows due to broncho-pneumonia are similar in character to those of the lobar type, except that they are not confined to one lobe, but are of an irregular or patchy distribution—often in the lower lung. Its X-ray appearance may simulate tuberculosis. The rapid change in plate findings, the fact that it is usually in a lower lobe, the history and clinical and laboratory findings make the differentiation.

The shadows due to influenza-pneumonia are not characteristic, and their distribution is variable. These shadows are of a hazy and mottled appearance, which may be localized to one or two bronchial branches, or may be very extensive. Here again,

the rapid change in plate findings, together with the history and clinical findings are essential for a diagnosis.

For some time after the active process has subsided, any of the pneumonias may leave changes in the lung which on plates give an increased density of the linear markings very similar to that seen in the chronic inflammatory conditions, such as bronchitis and asthma.

The plate in Fig. 28 was taken twelve days after the fever of a severe influenza had subsided, and illustrates early post-influenzal changes. Throughout both lung fields, the linear markings show a uniform increase in density which is fairly heavy. Very similar

Fig. 28. Post-influenzal changes. Increase in density of lung markings simulating a chronic inflammatory process.

plate findings may also be seen several weeks or months after an attack of either influenza or pneumonia.

In tuberculosis, in contradistinction to the pneumonias, the densities are uneven and are more frequently located in the upper lobes, and the findings are constant on repeated examination, even after relatively long intervals.

Miliary tuberculosis gives shadows of increased density due to pathology in the lung parenchyma which obscure, rather than follow, the triangular or fan-shaped arrangement of the bronchial branches. Myriads of small, fairly dense spots, each with a hazy halo, are scattered promiscuously, usually throughout both lungs. These hide the lung markings and give a hazy, stippled appearance which suggests a fine snowstorm on a gray day. The gen-

eral density may be a little heavier in the upper than in the lower lobes. The size of the flake-like spots may vary slightly in different cases, but is practically uniform in each individual case. There is usually little or no apparent increase in the density of the hilus shadows.

Figure 29 illustrates a case of miliary tuberculosis. Here the stippling is in small flake-like spots, each with a hazy halo, scattered very profusely throughout both lungs. A heavy, triangular shadow extends to the periphery in the left second and third interspaces and is covered by larger flakes. It is reasonable to assume that this area is the site of an earlier tuberculosis, from

Fig. 29. Miliary Tuberculcsis.

which the miliary dissemination took place. The patient died with the clinical picture of miliary tuberculosis a week after this plate was taken. An autopsy was not obtained. On screen examination, there is an increase in density, quite uniform throughout the area involved, often with a blotched or spotted appearance. Diaphragm excursion may be limited.

The shadows of miliary tuberculosis are rather typical on plates and their differentiation is often necessary. The plate, on the contrary, becomes of diagnostic importance because the clinical and physical findings are often absent or indefinite.

A few other lesions have a miliary distribution of their shadows on X-ray plates. They are not very likely, however, to be confused with miliary tuberculosis.

In advanced cases, the nodular type of metastatic carcinoma appears on plates in a miliary distribution so profuse that both lung fields are covered by rounded shadows due to the metastatic nodules. On the same plate, these shadows may vary in size from very small up to two centimeters in diameter, and in density from very faint up to the density of the heart shadow. There is a fuzzy or "Cotton-ball" appearance about the periphery of each spot which is quite characteristic.

Figure 30 illustrates a plate of metastatic carcinoma of the lungs. This patient had had a breast amputation for carcinoma some time before, with recurrence and evidence of lung metastases at the time the plate was taken. The plate shows all the characteristics of the shadows just described.

Fig. 30. Metastatic Carcinoma. Nodular Type.

The shadows due to sarcoma of the lungs are occasionally numerous enough to be considered of miliary distribution and be confused with miliary tuberculosis (113). More usually they appear on plates as one or several widely separated spots of variable size and density, the peripheral margins of which, in contradistinction to carcinoma, are sharply defined. The plate in Fig. 31 illustrates a case of the latter type of sarcoma. In this plate, one large and three smaller, fairly dense, rounded, sharply defined shadows are present on the right side. These are due to the sarcomatous nodules. The left lung is not involved.

The shadows of multiple gummata of the lungs are said to

have much the same appearance as sarcoma; here serological and therapeutic tests are needed.

The type of plate shadows appearing in the region of the lung parenchyma as an increased density, which veils the linear markings, but may or may not follow the triangular arrangement of the bronchial branches, is that due to pulmonary hemorrhage. In this type, a cloud-like shadow appears which may be localized or extensive, and which may vary somewhat in density in different parts of the cloud. Lung markings may or may not be seen, or, in places, may seem to show through this cloud of increased density. Variations due to hemorrhage depend not so much on the total quantity of blood lost, as on the position of

Fig. 31. Metastatic Sarcoma. (A) Areas representing metastatic nodules.

the blood retained in the bronchi, and also the amount and type of reaction which may have taken place in the lungs.

Figure 32 is the plate of a medical student, who without warning or previously recognized symptoms, had a severe hemorrhage, followed by a second hemorrhage the next day. The total quantity of blood lost was estimated as two quarts. A few tubercle bacilli were found. This plate, taken three days after the second hemorrhage, shows a heavy fan-shaped cloud of increased density covering the left hilum, and extending well to the periphery in the left first and second interspaces. Almost no lung markings are seen in this area. In the right hilum is a mass of rather heavy, rounded shadows. The findings elsewhere in both

lungs are practically negative. On fluoroscopy, there is simply an increased density, often very heavy, in the region of the hemorrhage.

A hemorrhage shadow may be quite similar to that of a pneumonic consolidation. Hemorrhage appears at once as a heavy shadow, and is less rapid in its subsequent change—here the history and clinical findings are of great value.

Still another type of increased density, due to tuberculosis, which on plates is in the region of the lung parenchyma, which blurs or obscures lung markings, and which may or may not follow the fan-shaped arrangement of the bronchial branches, is that due to healing processes such as scar tissue or fibrosis.

Fig. 32. Pulmonary Hemorrhage.

These shadows are of an even or "solid" density which may appear in localized streaks or bands, small spots or larger masses. The fan-shaped shadow of the healing tuberculous lesion becomes denser than in the more active stage. Its linear markings become more definite and a little wider. The stippling either disappears or changes to small, dense, even calcified spots, which, scattered through the area, add to its density. A small, healing, fan-shaped area may thus seem to be a small, almost solid mass, streak, or band on the plate.

Small cavities may be lost by contraction of the scar tissue, or obscured by the shadow of the thickened wall, giving the ap-

pearance of a smaller or larger dense spot as was pointed out under Fig. 25.

In marked cases of fibrosis, the shadow is a heavy density, often more or less irregular in outline, and in the degree of its density. Such a shadow may be dense enough to blot out all the lung markings in a lobe, the greater part of a lung, or parts of both lungs. The heart shadow may be pulled completely to one side or its outline lost in the general density. The shadows of the aorta and trachea are frequently pulled far to one side. The diaphragm may be drawn upward or made irregular in appearance. Even the chest wall may be contracted.

Figure 33 is shown as an example of marked fibroid phthisis

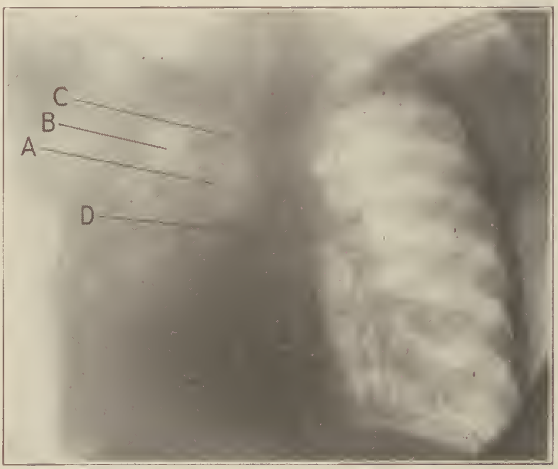


Fig. 33. Fibroid Phthisis. (a) Mass of heavy streaks and bands; (b) Oval, relatively clear, area; (c) Trachea; (d) Left Bronchus.

with distortion of the outlines of viscera. In this plate, there is a heavy density throughout the right side. It is practically solid in the lower half, obscuring the diaphragm and lung markings. Even bone density is lost except for parts of the ribs near the lateral margin. The heart shadow must lie within this solid density, for it is not made out elsewhere on the plate. In the upper half of the right lung the density is not so uniformly solid and rib outlines can be followed, though lung markings cannot. Just below the mid-portion of the clavicle and above the level of the third rib anteriorly (Fig. 33-a), the density is made up of a compact mass of heavy streaks and band-like shadows, none of

which can be traced for more than a short distance. These are a part of the fibrosis. An oval, relatively clear area (Fig. 33-b) lies in the first interspace and overlaps the first and second ribs anteriorly. This is probably due to partial pneumothorax. The shadow of the trachea (Fig. 33-c), is curved and lies to the right of the spinal margin from the second to the sixth dorsal vertebra. There the left bronochus (Fig. 33-d) crosses the spine shadow.

The left lung is relatively clear, although its linear markings are uniformly intensified, extend well to the periphery, and have a somewhat flaky or beaded appearance. The hilus shadows are fairly heavy, and several heavy bronchial branch shadows extend downward. The intercostal spaces are narrower on the right than on the left indicating some retraction of the chest wall.

This patient had a long history of cough, loss in weight, etc., and tubercle bacilli were found in the sputum. On screen examination, the shadow of such fibrosis is dense, even solid. The position of the distorted viscera can usually be made out. These shadows are to be differentiated from those which appear to occupy the lung field by virtue of their position, but which are due to pathology outside of the lung.

A uniformly increased density, covering the region of the lung field and varying from a faint haze to so dense a shadow that lung markings are obscured, may speak either for fluid in the pleural cavity, or for thickened pleura. If the shadow is faint, it is more often due to thickened pleura, as the fluid shadow is usually, though not always, heavy. Given a heavy shadow, if the contour of the chest wall on the affected side is contracted, as shown by narrowing of the intercostal spaces at the lateral margin, and if the mediastinal viscera are not displaced toward the opposite side, then the increased density is very probably due to thickened pleura. A displacement of the mediastinal viscera, en masse, toward the opposite side without narrowing of the intercostal spaces, speaks strongly for the increase in density being due to fluid. If the shadow is uniformly dense, the intercostal spaces narrowed, and the viscera as a whole pushed toward the opposite side, there is probably a combination of fluid and thickened pleura. This combination is not infrequently seen in tuberculosis, but may also be due to other causes, as empyema, or carcinoma of the pleura by direct extension.

Small amounts of free fluid obscure the costo-phrenic angle only, larger amounts extend higher in the pleural cavity, and hence obscure more of the lung field. The upper border of these fluid shadows has a definite outline, which curves upward at the outer or peripheral margin, unless the fluid so completely fills the chest that the upper border is lost under the clavicle and first rib. Exudative pleurisy, or hydrothorax, is so frequently associated with tuberculosis that, with such plate findings, it must always be kept in mind.

Figure 34 illustrates the plate findings of hydrothorax.

On the left, extending upward to the level of the second interspace anteriorly is a uniform, very dense shadow, with a well

Fig. 34. Hydrothorax.

defined, upper border, which curves upward at the outer margin. The diaphragm, left heart outline, and lung markings are hidden by it. The right border of the cardio-vascular shadow is seen farther to the right than normally. The lung markings which lie above this shadow, and also those of the right side are normal. These findings alone are not diagnostic for tuberculosis, but are suspicious for it.

On fluoroscopic examination, hydrothorax appears as a solid density which curves upward at the outer margin, and which is not movable on respiration or with change of position of the patient. (The fluid associated with certain tumors may be slowly

movable. If air is also present, the fluid is freely movable.) The relative position of small amounts of fluid in the chest may be determined by viewing the patient from different directions during the fluoroscopy. Increased density due to localized thickening of the pleura, may also frequently be an associated finding in tuberculosis.

If the angle of the diaphragm with the chest wall is clouded by a rather dense, more or less irregular, shadow, without the sharply defined upper margin due to fluid, although there may be an upward extension of the shadow at the lateral margin, a localized thickening of the pleura with adhesions to the diaphragm is definitely suggested. The dome of the diaphragm then appears flattened, or is irregular in outline, while the lateral portion is fixed. This fixation is best brought out by the limitation of the respiratory mobility of the diaphragm as seen on fluoroscopic examination. Fluid may obscure or limit the movements of the diaphragm but does not make it irregular.

A dense line a little inside of, and parallel to, the lateral thoracic wall is due to a localized pleural thickening, causing retraction of the lung and consequent small pneumothorax. The intercostal spaces in this region may also be narrowed. These findings are not uncommon in tuberculosis. Similar findings, a little larger in area, are associated with "walled off" pockets of fluid or pus occurring about the lung surface but not extending to the diaphragm. In such cases, however, the fluid level may often be made out.

If either a dense line, or a relatively small, fan-shaped, uniform density with base to hilum extends transversely across the lung field in a position corresponding to a lobe interspace, an interlobar pleural thickening is to be considered. Such findings accompany tuberculosis, as well as pneumonia, localized empyema, etc. Their position helps to differentiate them from the lines due to fibrosis or healing described above.

Very dense, irregular shadows which are well localized, or fairly extensive, and due to calcification of the pleura, may appear to occupy any part of the lung field by virtue of their position in the pleura. These shadows may follow, or be associated with, tuberculosis, an old empyema, a lung abscess, or, occasionally, old age. To determine their relative position stereoscopic plates are necessary.

On fluoroscopy, localized pleural thickening appears as a

heavy density in the region of the lung field. Its diagnosis and localization are aided by viewing the patient from different angles, and also by noting any limitation or irregularity in the excursion of the diaphragm.

Certain tumor masses, such as primary mediastinal malignancy and the infiltrating type of metastatic carcinoma, may give heavy shadows in the region of the lung field which involve the portion of the lung nearer the spine, instead of the peripheral portion as in tuberculosis. A mediastinal tumor casts a heavy, rounded shadow, usually in the upper portion of the lung field, and appears to be a single mass rather than a group of several smaller shadows. There is often considerable thickening of the lung markings about the tumor, but in contradistinction to tuberculosis, they decrease, rather than increase in density toward the periphery. If lying in the region of the aortic arch, a tumor is differentiated from an aneurysm by the fact that its margin is not so sharply outlined as the aortic shadow, and can nearly always be seen to lie in a different plane. On lateral plates, tumors usually extend into the posterior mediastinum; aneurysms extend anteriorly.

The infiltrative type of metastatic carcinoma might be mistaken for one of those relatively rare cases of basal tuberculosis. Carcinoma, however, is heaviest near the spine with a tendency to linear radiation of its markings, and a decrease in the general density toward the periphery; the opposite is true of tuberculosis. History and clinical and physical findings are again very valuable aids in diagnosis.

Figure 35 illustrates such a case. Here the density is very heavy and mass-like near the spine, from the arch of the aorta to the diaphragm, on the right side, but grows less dense as it extends outward, and practically ceases about half way to the periphery. This metastatic growth followed a breast amputation for carcinoma, and was confirmed at autopsy.

Decreased density in the region of the lung field may, on plates, be (a) localized or (b) generalized.

A. Localized. A localized or circumscribed decrease in density due to tuberculosis is that produced by cavity formation. Cavities are usually in upper lobes, may be single or multiple, and may vary in size from very small up to several centimeters in diameter. They are usually rounded in outline, with a margin which varies from very thin to very broad and dense. The area of de-

creased density due to the cavity may be more or less mottled depending upon the density of the lung markings in front or back of it. Cavities show an increase in density when they are filled with fluid. Occasionally, a definite fluid level with an overlying clear air space may be seen. A cavity is illustrated in Figures 25 and 26. Heise describes annular shadows very similar in appearance to true intrapulmonary cavities but which may change in size and shape rapidly. In some, a fluid level has been seen. He believes these are due to localized pneumothoracies and indicate pleural softening with rupture of the lung. They are rarely diagnosed clinically.

Cavities are not always made out on screen examination, espe-

Fig. 35. Metastatic Carcinoma. Infiltrative type.

cially if small. The surrounding density may be the only thing which attracts attention. On deep inspiration, an open cavity may grow clearer and a trifle larger. Fluid appears, as on plates, as a definite density and may be demonstrated by its mobility, if air is also present. Rotation of the patient aids in the localization of the cavity.

A bronchiectatic cavity or a lung abscess is most often in a lower lobe. The shadow of either is that of a rounded, hazy, or "pneumonic" area usually connected with the hilum by several heavy bronchial branches. This area is usually surrounded by a fairly well circumscribed zone in which the lung markings are much increased in density. Shadows of enlarged or dilated

bronchi speak strongly for bronchiectasis. These conditions are rarely due to tuberculosis.

B. Generalized. One type of generalized decrease in density, not a feature of tuberculosis, is briefly mentioned here for the sake of the differential diagnosis. In such cases, there is an increased transmission of the rays throughout the lung fields, due to an increase in the size of the air spaces of the lung. There is little change otherwise in the lung markings. A "barrel-shaped" chest with widened intercostal spaces and a depressed, flattened diaphragm are usually associated findings. Such X-ray findings speak strongly for emphysema.

A generalized decrease in density which, on plates, appears in the region of the lung field, and which is frequently found in cases of tuberculosis, is that due to pneumothorax. This condition often develops spontaneously in the course of a tuberculous process, or is produced artificially as a therapeutic measure.

On plates, pneumothorax gives an area of decreased density between the border of the collapsed lung and the lateral thoracic wall. The size of this area corresponds to the amount of air present, or conversely, the lung is compressed in proportion to the amount of air in the pleural cavity. The area between the lung margin and the chest wall is clear, and, of course, is devoid of lung markings. The compression of the lung may be slight, or so marked that the lung appears as a small mass about the hilum. If pleural adhesions exist, the compression of the lung is irregular, depending upon the size and position of the adhesive bands. The markings of the compressed lung appear to be increased in density because more compact, aside from any other lung pathology which may also be present.

Figure 36 is the plate of a patient in whom a spontaneous pneumothorax developed. Here, the left lung is compressed to about half its normal size. The space beyond the smooth, sharply defined, undulating lung margin is clear, and devoid of all lung markings. The lower half of the left lung is practically a solid density and some very dense markings are visible in the upper lobe. The increased density in the left lung is due to compression. The left diaphragm is depressed and flattened.

On the right side, the lung markings are uniformly intensified, somewhat beaded in appearance, and extend well to the periphery and into the apex.

If artificial pneumothorax is considered as a therapeutic

measure in a given case, the X-ray plate should always be studied before making the final decision. A plate is the best means of determining the real extent of the process, and, in cases of bilateral involvement, warns against compression of one lung if too great an area of the opposite side is involved. A plate aids in determining the best site for the injection, and also acts as a check on the results of the pneumothorax, by showing at once the amount of compression obtained, and, later, by showing the effect on the tuberculous process.

On fluoroscopy, the clear area of the pneumothorax is easily seen, while the area of the compressed lung is relatively dense.

Fig. 36. Pneumothorax.

Pleural adhesions, especially if fairly dense, are quite easily made out.

Plate findings due to hydro-pneumothorax are a combination of increased and decreased density since both fluid and air are present in this condition. The dense shadow of the fluid in such a case has a straight upper border, which is superimposed by the clear air space. Lung markings are wholly or partially obscured in the region of the fluid, are absent in the region of the air, and are compressed through the rest of the lung field. Pleural thickening may be present, which, of itself, or perhaps by adhesions which pull the lung slightly upward, may give the appearance of an upward outer curve of the otherwise level fluid surface.

Changes in the fluid level may be demonstrated by plates taken in different positions. With the patient horizontal, however, on postero-anterior plates or the reverse, the shadow of the fluid spreads out and obscures the clear air space.

Figure 37 is a plate, illustrating hydro-pneumothorax. In this plate the lower part of the left lung field and the left diaphragm are completely obscured by a dense shadow which extends upward as high as the level of the fourth rib anteriorly. At its upper margin this shadow is very sharply outlined, and forms a straight line transversely across the lung field from the spine to the lateral wall. The area above this line is clear due to the presence of air, and the lung markings are absent. The heart

Fig. 37. Hydro-pneumothorax

and aorta are pushed slightly to the right. The right lung markings are moderately intensified, and may be seen extending, practically uniformly, well toward the periphery. The right diaphragm is normal.

There are several small, dense shadows on the left side in the region of the lower lung field and upper abdomen, due to shell fragments. Clinically, the case was hemo-pneumo-thorax, due to war injury.

On fluoroscopic examination, hydro-pneumo-thorax presents a dense shadow due to the fluid, which is superimposed by a clear air space. The upper margin of the fluid is very freely movable

by slightly shaking the patient. Viewing the patient from different angles aids in determining the amount of fluid and air present, and their relative location in the chest.

It is hoped that the difficulties in the diagnosis of tuberculosis from the plate alone have been brought out. There are a few rather characteristic findings, such as shadows of calcified tuberculous glands, and of miliary tuberculosis, and some which are always very suggestive, such as the fan-shaped, hazy, rather heavily mottled shadows near the periphery in the upper lobes. But with all this it is to be emphasized that the relation of X-ray to the diagnosis of pulmonary tuberculosis is that of an adjunct, rather than the only means, or even the best method, of making the diagnosis.

C. B. R.

CHAPTER 18

MENSURATION, SUCCUSSION, VITAL CAPACITY, ETC.

(a) **Mensuration. Measurements of the Chest. Thoracometry.** Physical exploration by inspection may reveal that the chest as a whole is abnormally long from the neck down to the costal arch, or abnormally flat, or perhaps disproportioned, is not symmetrical, one half of the chest may bulge or protrude, or the other half be much retracted, is much smaller than the opposite half. These abnormalities or chest deformities may point to some disturbance in the thoracic cavity, perhaps, a tuberculous process; the retraction, to an old chronic fibroid phthisis, empyema, or pleurisy; an over distension of one half of the chest, to a vicarious emphysema, so that exact measurements are often necessary. In tuberculous individuals, even with apparently perfectly normal chests, measurements are often resorted to, to learn the amount of chest excursion. A normal, healthy individual usually gives a chest excursion anywhere from three to five or more inches. The individual in whom free in and out breathing is hampered by pulmonary disease often shows no chest expansion or, if any, but very little.

Chest measurements are usually undertaken as to circumference and diameter. The circumference of the thorax is generally measured with an ordinary tape measure graduated in centimeters and inches, usually on a level with the nipples in front and the angles of the scapulae behind with the arms outstretched or resting on the head. The circumference of the chest should be one-half that of the body length or height and the measurement should be taken while the subject is holding the breath. In old age, the ordinary chest measurements along the upper part diminish, and the lower chest circumference becomes greater. Normally the right chest is from 1 to 3 cm (one half to one inch) greater than the left. The diameter of the thorax, the long diameter, cranio-caudal measurement, the measurements from the clavicle to the base of the chest are very variable. It is scarcely possible to fix any one number as expressing its normal length; the appearance by inspection, however, often suffices.

The transverse diameter (the breadth), from the right axilla through to the left or vice versa in the upper part of the chest of the male adult, is about 25 cm or 10 inches, in the female about 23 cm, approximately 9 inches. The antero-posterior diameter, the sterno-vertebral (the depth), is about 19 cm or 7½ inches through the middle of the chest.

An asymmetrical chest, one in which there is on inspection a perceptible difference in the two sides, may best be measured by making a small ink mark over the midsternum on a level with the nipple and another in the centre of the spine on a line with the angles of the scapulae and measuring each half of the chest separately and carefully noting the difference. Various instruments for measuring chest deformities have been devised but few are in general use; a good steel tape line will answer all the necessary requirements.

In great chest deformities, as in scoliosis, kyphosis, or kyphoscoleosis, chest measurements must be undertaken at different levels along nearly horizontal lines and the different measurement of the two sides of the thorax carefully noted. The student is here again to be reminded that although there may be much chest deformity due to a distorted or twisted spine, tuberculous disease is extremely infrequently as accompanying disorder.

(b) **Succussion. Succussion Sounds or Murmurs. Splashing Sounds.** This method of physical exploration was well known to the ancients. It is closely described by Hippocrates and in the older writings it is frequently referred to as the Hippocratic succussion sound. Laennec, however, recognized it in association with pneumothorax, and gave us the first accurate description of this phenomenon.

Succussion is here briefly considered because it frequently is a symptom-complex of a pre-existing pulmonary tuberculous process. Succussion sounds can only be elicited if the fluid is free in the pleural cavity and then only when both air and fluid are within the chest cavity. Succussion sounds are produced by suddenly shaking the body of a tuberculous person suspected of having a pneumothorax, when if air and water are present in the pleural sac, a splashing sound becomes audible if the examiner's ear is placed directly against the chest. A stethoscope may be used but the splashing sounds do not become so pronounced. In exceptional instances, intrapleural succussion sounds may be recognized by the palpating hand when it is referred to as "suc-

cussion fremitus." A fairly good representation of this sign may be obtained by placing a rubber hot water bottle partially filled with water against the ear and shaking. An ordinary glass bottle or jug will answer the purpose fairly well.

(c) **Vital Capacity. Spirometry. Estimation of the vital capacity.**(32) The vital capacity of the lungs is the measurement of the greatest volume or quantity of air which is exhaled after the fullest inspiration followed by the most forced expiration. The total capacity is the vital capacity plus the quantity of air still remaining in the lungs after the most complete expiration. On ordinary inspiration and expiration, the "tidal air," amounts to about 500 ccm or 30 cubic inches. The "reserve" or "supplemental" air, that is, the amount of air which, after ordinary expiration, may be expelled by forced expiration is about 1500 ccm or 90 cubic inches. "Complemental air" or the amount of air which after ordinary inspiration may be inhaled by forced inspiration and the "residual air," the quantity of air which after forced expiration still remains in the lungs, are each estimated to be about 1500 ccm or 90 cubic inches. The vital capacity is variously given by different authors and while some limit it to 2500 ccm others estimate it as high as 3800 ccm. In a perfectly healthy man it amounts to about 3½ litre of air, approximately one gallon (231 cubic inches).

The vital capacity is proportional to body length, is influenced by sex, age, height of the individual, and it increases at the rate of 60 ccm for every cm of height above 155. The minimum vital capacity of a man of 155 cm (about 5 feet) is about 3000 ccm (or 180 cubic inches), and if 5 cm (2 inches) is added to body height, the vital capacity will be 3300 ccm, etc. As to age, the vital capacity increases about 160 ccm from the 15th to the 35th year and decreases about 500 ccm from the 40th to the 65th year. It also becomes less in the extremely aged and is less in the very young. The vital capacity is also increased in health as well as in disease by residence in a high altitude, that is, in mountainous countries. Sedentary occupations and defective nutrition lower and the opposite conditions increase the vital capacity.

As a rule, men between 20 and 40 years of age of medium height have a vital capacity of about 3600 ccm (230 to 240 cubic inches) and women about 2500 ccm (150 cubic inches) short people about 3000 and tall about 4000 ccm. Cornet places the minimum amount in healthy men as 1 to 20 and in women as 1

to 17, that is, 1 cm in body length (or 7/16th inch) in men is equal to 20 ccm (or about 4 cubic inches) of vital capacity, and in women 1 cm of body length (or 7/16th inch) to 17 ccm (or 3¾ cubic inches). All these figures vary considerably and can only be given approximately. They vary greatly as to age, the expiratory efforts and muscular force, fulness or emptiness of the stomach, etc. Any interference with the full or free expansibility of the lungs, such as diseases of the respiratory organs, will diminish the vital capacity. In advanced pulmonary tuberculosis accompanied by much tissue destruction, this may be reduced by one half and even more.

In beginning cases or early tuberculosis, the method is not always applicable as a diagnostic aid because the physical signs of palpation and percussion are positive long before there is a lowering of the vital capacity. Then again, the lessening of the vital capacity does not always run parallel to the amount of lung which is incapacitated, for in pleurisy with effusion when half of the lung is compressed the vital capacity does not drop to one-half because the functioning lung is doing compensatory work, which makes up somewhat for the loss encountered by the affected member. There is also an element of danger in spirometry in the active tuberculous because the forced inspiration and expiration may carry infectious material up into otherwise healthy portions of the lungs; then, there is also danger from hemorrhage by forcibly compressing or expanding a cavernous lung. Pleurisies, pericardial disease, emphysema, etc., all lower the vital capacity, and not only the diseases of the intrathoraic organs but muscular weakness may lower it.

Vital capacity, like blood pressure observations, can not be gauged by a single test; in fact, the first experiment is usually wholly unreliable because the patient has not learned how to breathe. It is only after repeated attempts that one succeeds in securing reliable data as to the vital capacity. If the free excursion of the lungs is prevented, that is, when breathing is accompanied by pain, when respirations are abnormally frequent, or when the patient is weak, vital capacity is not available as a diagnostic measure. In the beginning stage of suspected pulmonary tuberculosis, when the process is still quiescent, is not very active, is of the slow or proliferative type, the patient is not yet showing any or perhaps little loss in weight, has few clinical symptoms, the diagnosis not quite definite, the low vital capacity

in an individual when compared to height, sex, weight may become a valuable aid as a diagnostic measure, whilst in the actively tuberculous with positive physical findings and clinical picture, vital capacity can be of no value because here the diagnosis can be definitely made by physical examinations, besides, it may become a source of grave danger.

CHAPTER 19

THE CARE OF THE TUBERCULOUS

The Treatment of Pulmonary Tuberculosis. General Therapy, Ambulatory, Home, Sanatorium, Dietetic-hygienic, Medical, Tuberculin, etc.

General Consideration. Health and Disease. (20)

A human being is said to be in perfect health when there is perfect co-ordination of all the organs and tissues of the body, no functional disturbances; the direct opposite of this state is known as disease. Disease as we know it today is not, as was believed by the ancients, a visitation from heaven which must be borne with patience, nor an infliction from an evil spirit. It is brought about by our mode of living, our environments, our social relationship, and more than that it is the direct result of the violation of inflexible, inalterable and immutable laws which have been violated by the afflicted individual. In no other disease can this, the violation of these laws, be traced back to a period of transgression as in pulmonary tuberculosis, if we only persist in a diligent search.

Diseases have been variously classified as to duration. We speak of acute diseases when the onset is sudden, as in scarlet fever, measles, small pox, etc., and the duration is short; subacute when the onset is somewhat protracted, as in pleurisy, and chronic when the disorder is drawn out over more or less long periods, as is generally observed in syphilis, leprosy or tuberculosis.

We also speak of pulmonary tuberculosis, the disease now under consideration, as the most chronic of all, and we say that it is primarily a house or home disease, which it is in more than one sense. We all know that the primary infection, in by far the greater number of cases, is brought about in the home, being transmitted from the tuberculous adult to the child, that subsequently the disease is fostered in the home, and that the actively diseased individual constantly frequents the home. It seems that throughout the whole period of the affliction he is wedded to the home, is anxious to become well but only in the home.

Tuberculosis is spoken of as a preventable, (21) a curable disease, that the infection could have been prevented but was not, that even if once the infection is followed by manifest disorder it is still curable, provided the disorder has not existed too long and the damage inflicted is not too great. Pulmonary tuberculosis, as has been stated, is a chronic disorder, a scourge, a plague, the mortality and morbidity exceeding that of all other diseases.

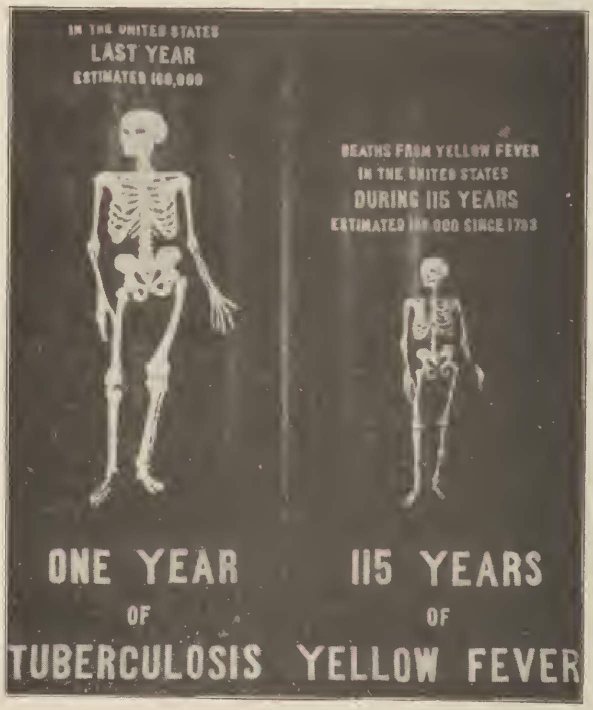


Fig. 38. Comparing the death rate from tuberculosis in the U. S. in one year with that of yellow fever in 115 years. (From the U. S. Public Health and Marine Hospital Service. Tuberculosis Exhibit, 1908.)

In the United States more than 160,000 (1908) succumb annually. and more than 800,000 are constantly sickened from this disorder. (The U. S. Census for 1920 shows the tuberculosis mortality to be 114 per 100,000, an unusually low death rate.) In the city of Chicago the annual mortality from tuberculosis is, in round numbers, approximately 4000, with a constant morbidity of more than 20,000. It is most frequently a disease of young or middle life, exacting in the latter its greatest toll at a time when the

earning capacity should be at its best and in the former at that period when the afflicted youth should be in a position to earn or at least to repay either to his parents or to his benefactors some of the interest accrued on the amount of money which was necessarily expended for his care, schooling, teaching, clothing and raising, etc.

Sources of Infection. If, as so often has been stated, everybody has at some time become infected, that in a certain proportion only of those infected the disease becomes manifest, that the problem has become a family or home infection, whence the propitious source of all this disorder? Close observation has demonstrated definitely that there are three chief sources—namely:

(1) Infection from the unknown tuberculous. We will find in many homes individuals suffering from so-called chronic bronchitis, chronic catarrah, from asthma, from emphysema, etc. In these, owing to the generally good physical appearance, tuberculosis is not suspected and many a good and kind old grandfather or grandmother suffering from these disorders, upon close examination, will be found afflicted with chronic pulmonary tuberculosis, and being unfortunately in constant companionship with the infants or small children in the household, will often be the source of a tuberculous infection. Again, a hired person about the home, either male or female, may be suffering every winter from a slight cough, and here also, owing to the healthy appearance, tuberculosis is not suspected. Often parents, either the father or the mother, have a slight cough or cold, especially if the onset is slow, causing little or no disturbance, general appearance is good, there is no loss in weight, tuberculosis is not suspected and even if a diagnosis of pulmonary tuberculosis has been made, it is not believed and the given instructions are not heeded. In such homes the disease is very often spread to all the young members of the household.

(2) As a second source of tuberculous infection and disease are the known tuberculous. First those living and belonging to the household, who are known to be suffering from pulmonary tuberculosis, but are careless about their person, do not keep clean, do not take proper care of their sputum, insist upon the freedom of the house, upon eating with the family at the family table and in other ways insistent on doing as they please about the home; they are a most prolific source of tuberculous dissemination.

Second: the homeless consumptive. A kind hearted sister or brother takes the homeless member of the family into the home, gladly provides a willing shelter, and themselves having families, expose their small children to a positive infection. Many a homeless and drunken father or a shiftless mother, being taken into the home of a dutiful and loving daughter has often repaid this act of kindness and duty by making the little ones of the household tuberculous.

(3) The third great source of tuberculosis is the discharged sanatorium patient. It is greatly to be deplored that in many instances the sanatorium patients leave these institutions with the firm belief, or at least make themselves believe, that a cure has been effected, return to their former homes or to the homes of acquaintances or friends to which they have free access, and now become the source of continuous infection. Often this unfortunate class of individuals is unjustly condemned for the injury which it has done and for which it was not at fault. Had these individuals been taught while at the sanatorium that the disease is not cured—that the process is only arrested, that at any time, if indiscreet, the disease may break out again and perhaps many times more active than the first, they would perhaps have been more careful and would have tried at least to avoid the spreading of tuberculosis.

The General Care of the Tuberculous. In caring for the tuberculous three classes or groups of consumptives are usually encountered: (1) the ambulatory, which comprises generally the first stage cases; (2) the bedfast or home cases, second and third stage, and (3) the sanatorium cases, consisting of some first stage, usually second and third stage cases.

(1) **The Ambulatory Cases.** These are usually under the care of the family physician. These patients generally consult the doctor, chiefly at the office, and it is this class which we so frequently see at the dispensaries. In these beginning cases, particularly when diagnosis has been positively made, it becomes the physician's duty to instruct the patient, truthfully, tell him what he has and teach him how to live to bring about an arrest of his tuberculous process. Many of these patients as a rule belong to the poorer classes, depending upon their daily labor for the support of themselves and their families. If the work is not too strenuous, they may be allowed to continue their vocation; if, however, it is very laborious, then lighter employment must be

secured. They must be incessantly encouraged to spend much of their time out of doors, rest a great deal out in the open, live in the outskirts of the city rather than in the congested districts, avoiding as much as possible much walking, observing the strictest rules of cleanliness and the proper hygiene about themselves and their homes or rooms, avoiding all excitement, being most abstemious in their habits, desist from the use of whiskey, wine, beer or tobacco, and be regular about their diet which must be good, wholesome and nutritious. The sleeping quarters and the sleeping rooms must be well ventilated, the windows open day and night. They must be told to remember at all times that the same good Lord who made the day air also made the night air, and that they must permit it to come freely into their houses and homes. These patients must be told that they must sleep alone and that the freedom of the house is only permissible if the case is a closed one and no tubercle bacilli have been found in the sputum. If, however, bacilli are present and the case then is an open one, they must spend most of their time in their rooms if they be not out in the open. If such individuals cough and expectorate, they must be taught how to take care of the sputum so as to avoid as much as possible a spreading of the disease. They must be told frankly that in their association with their fellow workers while exercising proper precautions, they are in no way a menace and they must be greatly encouraged to continue in their daily work. This tends to take the mind off their troubles and to bring ease of mind. As these patients usually at the very beginning of the disease consult the family doctor, he should evince the greatest interest in the case; this has a most favorable influence and a strong tendency to lessen fear in these beginning cases of pulmonary tuberculosis. While under the family physician's care, if the tuberculous process does not become promptly arrested, they sooner or later drift into one of the other two classes—either the home or bedfast or the sanatorium cases.

(2) **The Bedfast, the Home or House Cases.** This comprises both the second and third stage cases. In tuberculosis the home treatment is the most important of all, and at this stage the old family doctor is again looked to for advice. From the very beginning of the disease, from incipiency and up to the far advanced stage, even to exitus, he is asked to minister to this unfortunate class and his opinion usually carries great weight. Long before the case becomes bedfast the doctor, if he wishes

to do his full duty, should suggest sanatorium care. He must explain the method in use at such institutions, how these patients are being cared for and how by this method of treatment the disease becomes arrested. These patients at first are usually very much adverse to going and he must make clear to them what a sanatorium is. As about 9/10 of all pulmonary tuberculous individuals are not able, willing nor inclined to go to the sanatorium, the greater number will continue to be in the care of the family doctor, that is at the homes; this now becomes a most serious educational problem. From the very beginning these patients must be taught how to get well, how to make their surroundings safe for themselves as well as for others, particularly in the open cases. The sleeping rooms or preferably sleeping porches must be so situated that they can be readily aired and ventilated. A most desirable room is one with a southeast exposure, there must be little drapings or furnishings in such quarters and the bed must be comfortable and have sufficient coverings. Body cleanliness, bathing and sponging are most essential—if much fever and rapid pulse, absolute rest and quiet must be instituted. The diet should be wholesome and well cooked, bowels kept soluble and such symptoms as from time to time arise must be met by proper medication. Living in the open with much sunlight should be encouraged.

(3) **The Sanatorium Cases.** Considering the extremely great frequency of pulmonary tuberculous disease, there remains after all only a very small percentage, about 1/10, of all the afflicted who wish or are willing and anxious to go to a sanatorium—by far the greater number preferring to take the treatment at the home under the care of the old family doctor; nevertheless, the family doctor, as has been already stated, should in every instance at the very beginning, suggest the sanatorium. This, on the part of the doctor, often requires a great deal of tact, and owing to the patient's peculiar psychic state, becoming easily irritated, even the allusion to a sanatorium may be sufficient to cause a change of medical attendant.

The physician from the very onset must impress upon the patient's mind the importance of knowing that a tuberculosis sanatorium is in no sense a hospital but only a school in which the afflicted person is taught how to get well and how to remain so. It may be stated here parenthetically that it is true that a tuberculosis sanatorium is a school, but it is in no sense an ordinary school. It is strictly a reform school, in every way and only if the strictest rules are lived up to and the most rigid

discipline instituted, will the patient get well; this requires on the patient's part the most passive obedience.

The patient should next be informed of the simple fact that at the sanatorium he is under constant observation—daily and hourly—that there he will be taught the conditions of his disease, that a strict record of his case is kept at all times, that he is submitted to a periodic examination, as his case may require, that when it is deemed advisable he will be forced to take the necessary rest, and that his presence at the sanatorium brings about a change of surroundings, and as he notices and observes the steady improvement in others he becomes gradually more encouraged about his own case and that by these favorable environments he becomes more and more obedient, more so than at home, where he wants to do as he pleases. Throughout his stay at the sanatorium he will then become more and more impressed with the truism that "no tuberculously diseased individual ever became well or had his tuberculous process arrested, who wanted his own way." Observe the regularity in the diet, the mode of living, the medication, the treatment, the very atmosphere at a sanatorium. They are all factors which lead to an arrest of the tuberculous process.

The question is often asked, "How long should a tuberculously diseased person remain at the sanatorium?" Until the process is surely arrested, until he has learned his lessons well and has come to believe that in the future and at all times he must practice vigilance, that any indiscretion on his part may reactivate his old trouble and that when he returns to his home he must be willing and determined to lead a regulated, orderly and abstentious life. The old adage, "Once tuberculous always tuberculous" perhaps may not be literally true; nevertheless, the healed or arrested tuberculous individual should shape his mode of living as though it were true.

We have observed that when the tuberculously afflicted individual first begins to sicken of the disease he consults his family doctor, and now upon his return from the sanatorium to his home, we observe again that the old family doctor is the first to be consulted; hence, it becomes the doctor's duty to constantly remind the patient not to forget his institutional training. This is most important. The returned sanatorium patient needs much encouragement and this should be freely allotted to him by all, the physician, the nurse, the social worker, fellow workmen and

his associates. The aim of these patients should be to maintain their restored health and in this the family doctor will be the most important councilor. The next important problem for the patient to consider is his ability to again provide for himself and for his family. Here again the family doctor must advise. This necessitates work in which he needs the encouragement and support of every one.

The tuberculous patient returned from the sanatorium usually has been well trained, knows a great deal about tuberculosis, about the difference between infection and disease, and is in general a good disciple of the anti-tuberculosis gospel. He generally desires to remain much out of doors, preaches the doctrine of out-of-door life, the use of wholesome, good food and much rest, of diet, of retiring early and sleeping late. He generally submits every thirty days to a re-examination, knows well the meaning of a rapid pulse, watches closely the temperature curve, leads a quiet and unexcitable life, avoids excesses of all kinds and aims in general to maintain his body resistance, all of which, however, often has a strong tendency to make these individuals lazy, yes, even sluggards, and here again the physician has a great task to perform in preventing these returned sanatorium patients from becoming chronic sitters. Too much lounging about the house must be discouraged. The patient must become from day to day more reliant upon himself just as he was before he was made sick.

Tuberculosis a Threefold Problem. Pulmonary tuberculosis as we see it is a threefold problem, a social, and economic and a medical one, and to bring about healing, relief and betterment of surroundings it is required that the whole community become interested. Being a community question all have a duty to perform, not only the physician and perhaps the nurse but the community as well. The physician's duties have already been described somewhat in detail, namely, care of the patient from the medical standpoint, advice as to rest, order, adequate and wholesome nourishment, regulation of the home life and treatment of the symptoms as they arise. Then there is the duty of the attendant or nurse as to the housing conditions, seeing that the sleeping rooms and sleeping quarters are properly ventilated, that for the taking of food the open tuberculous is supplied with individual dishes, knives, forks and spoons, which are to be kept entirely separate from those in use by the other members of the household. The nurse must further teach the patient how to

take care of the sputum, how to gather and to destroy it so as to guard the spreading of the disease, how to keep a record of the daily doings, to regulate the time for visitors, she must encourage frequent bathing and sponging, in fact, everything must be done to keep up and increase body immunity.

A still greater duty to perform is that of the community at large towards the tuberculous. The tuberculously afflicted individual, in whatever walk of life, needs much encouragement, needs assistance, and he must be made to feel that because of his affliction he is not treated as an outcast but that the whole community is greatly interested in his welfare. If he is in poor or needy circumstances and is willing to go to a sanatorium, provisions must be made by the community to provide what is necessary for the care and comfort of his family while he is away from home and taking the cure. This can only be done by the community as a whole; however, charity organizations, welfare workers, insurance companies and associations, and philanthropists are generally the proper source for relief and they will take care of this class of cases if only their attention is directed toward them. If the patient is not willing to go to a sanatorium and wishes to take the cure at the home, then again these various organized bodies must come to the relief and suitable sleeping porches, blankets, bedding, food and care be provided. In every individual instance either at the sanatorium or at the home everything must be done with the sole object of returning the tuberculous back to the community as a fairly capable working unit.

The Treatment of the Tuberculous. General Therapy.(50) Tuberculosis is a constitutional disturbance in which both the body and mind require special care hence we must institute both a somatic and a psychic therapy. From the very beginning of the treatment our aim must be to endeavor to bring the diseased individual back to normal. In almost every case of pulmonary tuberculosis, if the process is not too extensive often even in cases of more or less extensive involvement, an arrest and even an apparent cure may be brought about if in the treatment we can correlate six well known and well established factors, namely, (1) ease of mind; (2) wholesome fresh air; (3) rest and appropriate exercise; (4) the diet, the dietetic-hygienic treatment; (5)

time; (6) obedience. These six factors and no others are always necessary to bring about in a tuberculous individual an arrest of the diseased process—all depending upon their proper harmonizing. If either one is wanting our efforts will be of no purpose. (See illustration, page 543.)

(1) **The Psychic Factor. Contentment or Ease of Mind.** Education. From the very beginning it must be our aim to educate the patient and to explain to him the nature of the disease. We must speak the truth, we must not tell him that he is simply suffering from a little catarrh, when we positively know that he has active tuberculous disease. On the other hand, we must not frighten him but on the contrary convince him that although he is suffering from pulmonary tuberculosis he is not condemned to die and that by having confidence in his physician he must follow his instructions to the letter and in that way he will get well. In patients suffering from far advanced pulmonary tuberculosis it is above all essential that we ease or relieve the despondent mind even to a point of shading the truth, because here speaking the whole truth would be cruel, nay, criminal. From the long drawn out, chronic condition of this disorder many patients become quite hopeful and buoyant even to the last, and here it becomes the physician's imperative duty to encourage this buoyancy. Some tuberculous patients become very despondent when they observe their helpless condition and the conditions about the home, about the care of their families, etc. Here ease of mind is often most helpful if aid is offered by the community, the charity organizations and allied bodies, knowing that suitable measures assure prompt relief and much comfort to his dependents. This also applies to individuals who are taking the cure at a sanatorium where either the husband or the wife should be assured that during their stay at the institution and while they are taking the cure, the other members of the family, those remaining at home, will not want for anything.

This principle of "Contentment," however, finds its most appropriate application in those cases for which it has originally been advised, namely, for the youth suffering from pulmonary tuberculosis and who is taking the cure in a more equable climate, perhaps thousands of miles away from home where owing to the want of "ease of mind," by far the greater number become victims

of nostalgia, become homesick and succumb early to the disease. If, for instance, a tuberculosis patient be advised to go away from home, that a change of climate be desired to effect a cure then one or more of those who are near and dear must make the sacrifice and accompany the sick member of the family. Do not send him or her alone out into a strange land, perhaps many hundreds of miles away from home, from friends, from those who are near and dear when the thoughts and the mind of these unfortunates during the long nights of vigil are constantly about the home, the friends, the dear ones, the always earnest desire and longing to be with the loved ones. Admitting that climate is a factor in hastening the cure, it should be weighed well before the afflicted person is sent away—only when the physician has the positive assurance that the patient's mind is at ease should this be allowed. Since the establishing in every community of tuberculosis sanatoria which are easily accessible and not too far from the homes of the afflicted, the leaving of home to go many hundreds of miles can be discouraged, as these institutions being near to the homes can readily be visited by relatives and friends on proper visiting days. The patients look forward to these days, and being encouraged in this way "ease of mind" is brought about.

(2) **Wholesome Fresh Air.** The second necessary factor is out-of-door living with plenty of fresh air. It is not necessary that the consumptive, in order to take the cure, must go up into the mountains or live in the valleys, in either a high or a low altitude, it is most essential that wherever he takes the cure that the air is pure, is free from dust and dirt and free from obnoxious gases or coal dust; nor must the humidity in the air be too great. The air must be wholesome and fresh, but it is not necessary that it be icy cold, for warm air will do just as well and perhaps better, provided it is pure. The air warmed by the sun's ray is most ideal, and inhaling pure air while there is much sunshine is most valuable—that is, combining a mild sunbath while taking pure air and remaining much out in the open, away from dusty air, free from obnoxious gases, where there is no noise and no excitement, out in the open, winter and summer, is the ideal way to take the open air treatment.

(3) **Rest and Appropriate Exercise.** In the treatment of the various conditions in pulmonary tuberculosis it is necessary that at times we apply absolute rest and that we institute when necessary gradual and graduated exercises with much rest in the open and out of door sleeping. With much rest in bed, or graduated rest like graduated exercise, often very much can be accomplished. As a rule febrile cases require absolute rest while in afebrile cases graduated and gradually active exercises favor autoinoculation. Absolute rest, only during the active, febrile stage, is the treatment in this the first stage. In the second and third stages if afebrile regulated exercise and a physical and an occupational therapy. If the pulse is rapid, the temperature about 100 or more, during the 24 hours, then absolute body rest must be instituted. The patient should be placed in the recumbent position, in bed, and must remain lying mainly on the involved side until his temperature approximates the normal and remains normal for a week, perhaps varying in the afternoon to about 99. It can not be too earnestly emphasized that under absolute rest we understand that the patient must remain in bed the whole 24 hours of each day. He must take his meals in bed and for toilet purposes he must make use of the bed pan. A patient suffering from active pulmonary tuberculosis, with a fast pulse and high temperature, must be treated just like one who is suffering from an acute disease like typhoid, pneumonia, etc., and absolute rest must strictly be insisted upon. For a patient with a daily temperature above 100 and rapid pulse, a 15 minute stay out of bed would completely inhibit or negatate the good work which was accomplished by remaining in bed the other 23¾ hours. Only the most strict adherence to this rule of absolute rest leads to success. If, in the treatment of our home cases we could institute the principles of absolute rest, often for weeks or months, so admirably enforced in a sanatorium, much better results would be obtained and many more cases would get well. If in a given case the temperature approaches the normal and the pulse wave begins to lessen, then graduated rest can be directed, but always bearing in mind that the moment fever again becomes high we must at once return to the absolute rest treatment. Rest lowers fever and activity, reduces the rate of breathing towards the normal, increases the appetite, inhibits night sweats, exerts a favor-

able influence upon the skin, lessens the cough and has a most salutary tendency on the condition of the lungs. In some pulmonary cases showing much activity, rapid pulse and high fever, often much good can be accomplished if the tuberculous individual, whilst at rest, is placed with the head and chest on a near level, slightly elevating the abdomen and lower extremities, when by means of autotransfusion and passive hyperemia the tuberculous processes seem to show a greater healing tendency. With graduated rest, graduated exercise go hand in hand. If the patient manifests much improvement after beneficent rest, then sitting up for ¼ to ½ hour is advisable; if no fever follows the time out of bed may gradually be extended and perhaps it might also be well to exercise slightly, taking a little walk about the room, always observing closely above all the temperature and the pulse. The patient should be told to rest a great deal in the open and to walk very little or not at all. The notion common among the public that the tuberculously diseased person should take long walks in the open is most pernicious and more damage than good has resulted from this practice. Walk very little, only as the case may require, and here as in all other diseases we must individualize. Tuberculous patients who are improving, and the conditions seeming to approach the normal should walk before meals and rest after. Rest from one to two hours after each meal and take a short walk before meals. Here again we must be cautious and careful in our advice, because too much rest is just as injurious as is too much exercise. We must aim to strike a happy medium. Then again, the amount of rest and exercise ought to be regulated according to the seasons, more rest in summer with less exercise and in winter the reverse — a trifle more exercise and perhaps less rest. The temperature curve and the pulse rate must be our guide, and from putting the patient to doing a little light work and gradually changing to heavier, or perhaps first from light sports, croquet, etc., gradually to tennis, golf, etc., we often observe a steady improvement and so the tuberculously diseased individual not infrequently finds himself gradually restored as a former working unit being again capable of resuming his previous vocation. Last the patient should continually bear in mind that it is indeed a long

drawn out process and that only obedience and perseverance on his part will lead to a triumphant issue.[1]

(4) **Diet.** (A) **The Dietetic-Hygienic Treatment.** Diet is the basis, is the fundation of all treatment in tuberculosis, or more properly the dietetic-hygienic, as first applied in the middle of the last century by Brehmer (1859) and later by Dettweiler. As the digestive apparatus in the normal is of the greatest importance in maintaining the body in a healthy condition, so it becomes the first and foremost in the diseased condition, to aid in bringing the body back to health. In the dietetic care of the tuberculous no special food is prescribed. The rule is to give what the stomach can easily digest, to give it with regularity, but always to save the stomach. Give the stomach sufficient rest; do not overeat but rather undereat; eat only such food as the system craves and then only in moderation and do not recommend foods which are most obnoxious to the patient. Eat with great regularity and at meal time only, allowing the stomach sufficient rest between the meals. The stomach like every other organ of the body partakes of the general tuberculous disturbance and if we institute general body rest in the treatment of tuberculous disease, why not stomach rest? Eat nothing between meals; if, however, the meals are very small, eating between meals may be advantageous. In health, we usually give

[1]The great importance of absolute body rest in the treatment of pulmonary tuberculosis can not be sufficiently emphasized. Even with the instituting of most complete rest, it is after all only relative. Where complete rest is possible it often requires years to effect a cure, how much longer time must then be required to arrest a tuberculous process in which complete rest is a physical impossibility. A child suffering from tuberculosis of the spine is put upon a Bradford frame and securely strapped; the parts are put to absolute rest and even here it often requires after complete immobilization years to effect a cure. This inhibition of motion is readily applicable in most cases of tuberculous disease of bones and joints, but it cannot be applied in disease of the kidneys, larynx, lungs, etc. In lung tuberculosis, the inhibition of pulmonary motion is a physical impossibility, and yet we must recommend absolute rest for the cure of all active cases.

A healthy individual under ordinary conditions breathes about 20 times a minute, 1,200 in an hour, 28,800 in a day, 864,000 in a month, and 10,368,000 in a year, and even here we observe that deep or full compensatory breathing is occasionally required. The pulmonary tuberculous patient during the period of his activity, often greatly increases his breathing cycles by additional thousands each day, his number of full or deep breaths is also increased, and the toxins circulating in the blood stream irritate the circulatory apparatus, causing increased cardiac action; this all lends additional motion throughout the pulmonary tissue, thus decreasing the amount of rest; besides, emotion, excitement, nervousness, are all additional factors in this lessening of rest.

As it is a physical impossibility in the treatment of pulmonary tuberculosis, to put the lung absolutely at rest, we must aim to bring about at least a maximum amount of rest with a minimum amount of exercise or lung excursion. At the same time, all such factors which tend to disturb this necessary rest must be removed as much as possible.

We frequently observe, in a child who is suffering from joint disease, that its parents are perfectly contented when told that with absolute rest the child can make a complete recovery, although this may take years, but when we are treating a young individual suffering from pulmonary tuberculosis and in whom pulmonary rest can only be relatively instituted, parents generally expect a cure in a few months. The only rational method for the cure of pulmonary tuberculosis, if the process is active, is to insist upon absolute body rest, in the recumbent position with the patient lying on the affected side, thus controlling the breathing motions by giving the lungs the maximum amount of rest with the minimum amount of motion consistent with life. To accomplish this fully we must, in addition, insist upon very little or no talking, the control of coughing and the avoidance of all excitement or emotional disturbances.

the stomach an hour or so rest after having digested a full meal, and this generally requires about 3 to 3½ hours, why should we not give sufficient rest to a weakened and irritated stomach, which is usually an accompaniment in the tuberculous? Forced feeding or stuffing so much in vogue a few decades ago is with our present knowledge concerning the treatment of this disease no longer tenable. Besides, it is harmful as the stomach is wholly incapable of digesting a large quantity of food at a single ingestion.

The tuberculous patient who under a properly regulated diet gains slowly and steadily in weight presages a favorable prognosis; a rapid gain is not a good sign as this does not indicate energy but simply fat and water. Our aim in the treatment is not to make the afflicted person fat, as body fat or increase in weight alone usually consists only of water, but to produce strength of muscle, healthy blood and energy—not quantity of cells, but quality. The best, not the greatest, must be our wish and aim and for the achieving of this, a well regulated diet, well prepared and wholesome, must be secured, preferably a mixed one consisting of animal and vegetable food with sufficient heat units or calories to maintain the patient's present condition and at the same time secure a steady and gradual increase of his body cells. This is done by building up and increasing his body strength. In order to assure this the various foodstuffs consisting of proteins, fats, carbohydrates, etc., must contain a sufficient number of heat units or calories to supply the output of heat and to equalize the body metabolism. Proteins, fats and carbohydrates each have food values estimated in calories and a sufficient number of each is necessary for the proper maintenance of life. Protein is an absolute essential for tissue building and repair; heat and energy of the body can be developed from the carbohydrates and fats. In food values 1 gram of protein equals 4, 1 gram of fat equals 9 and 1 gram of carbohydrate equals 3½ to 4 heat units or calories. (See Chapter 39.) The requirements of a healthy individual's intake in proteins, fats and carbohydrates (an individual weighing about 150 pounds) while at rest will approximate very close to 3000 calories per day; for light work it may be increased to 3500 or 3600 and this will require about 120 grams of protein—4 ounces equal 480 calories, 60 grams of fat—2 ounces equal 540 calories, and 500 grams carbohydrate—17 ounces equal 2000 calories, a total of 3020 calories.

The tuberculous individual while taking the rest cure would require daily about 3000 calories, whereas a man doing heavy manual labor would require about 4000 or about 196 grams of protein (or 6½ ounces) equal 784; 126 grams of fat (or 4 ounces) equal 1134 and 522 grams of carbohydrate (or 17½ ounces) equal 2088, equal almost 4000 calories. The necessary foods should consist of meats, fish, poultry, eggs, cheese, eggnogs, butter, fats, and oils,(71) milk, whey, koumiss, `bread, potatoes, puddings, fruit, nuts, vegetables like cabbage, spinach, etc. Aim to reduce the output rather than the intake. Further, throughout the treatment we must strictly inhibit the use of alcoholic beverages and the use of tobacco, and limit the intake of coffee or tea. In order to outline a proper dietetic course for the tuberculous and to estimate the necessary food values we ought to make a thorough study of each patient's case. Every patient's requirements in proteins, carbohydrates and fats should be carefully ascertained so that the daily amount of intake does not exceed the individual's tolerance. Proteins above all should constitute a fair proportion of this advised diet, but not more than the organism can take care of. In many instances in order to supply the immediate wants of the economy the nitrogenous food must be increased. This, however, requires often a great deal of good judgment not to go beyond the patient's protein tolerance. Nitrogenous food intake relative to tissue metabolism has received much attention particularly from dietitians, but not the over intake, that is, that amount over and above the necessary tissue waste and repair. Too much protein intake causes either too much renal work or the unutilized nitrogenous material may be the cause of intestinal putrefaction and disturbances followed by additional toxin absorption; hence a wholesome and generous diet, consisting of carbohydrates, fats and proteins, in such varying proportions, in amounts sufficient and suitable, just like the patient enjoyed when he was still in perfect health, will be found the most serviceable and appropriate in his diseased condition. It should be allowed with the same regularity and prescribed intervals as in health, at meal time only, giving nothing between meals. Thanks to a better understanding of the underlying principles which govern the processes of this disease, the pendulum has swung back and forced or over feeding and stuffing is now obsolete, but we must be guarded not to go to the opposite extreme and advocate underfeeding as the panacea in this tuberculosis problem.

In the dietetic treatment particular attention must be paid to the conservation of the digestive integrity of the stomach and intestinal tract, constantly bearing in mind that in health the gastro-intestinal organs offer the greatest protection against diseases of the lungs and that the digestive apparatus also offers the first aid and is chiefly to be relied upon when the lungs are diseased.

Therapeutically then, it may be stated that an increase of nitrogenous material in the diet of the tuberculous may be of undoubted value, but we must not lose sight of the fact that this must be kept within the patient's albumin tolerance, that a high protein diet over and above that used in tissue metabolism may cause in the intestines a rapid bacterial increase, intestinal putrefaction with toxin production and toxin absorption, and that this intestinal autointoxication is usually proportional to the nitrogenous intake.

(B) **Zomotherapy. From Zomos, Meat Juice.**(125) (139) The treatment of pulmonary tuberculosis by means of the freshly expressed meat juice. A strictly protein therapy. It has long been observed that meat is almost indispensable in the treatment of pulmonary tuberculosis and that raw meat or preferably raw meat juice is a most readily digestible and assimilable nutriment, a nerve stimulant, and that it is particularly indicated for the anemic, the convalescent, the undernourished, the chlorotic and the hemorrhagic and that with a patient in bed on a raw meat diet the temperature often becomes reduced. For a weak stomach the expressed meat juice is most suitable, particularly the muscular plasma of horse meat a most excellent therapeutic agent, being rich in both haemoglobin and glycogen. This expressed juice found much favor and soon became known as Horsine and it was but a step from taking freshly expressed meat juice to that of fresh blood, and the drinking of beeves blood for a time became quite popular. The drinking of fresh meat juice for the cure of pulmonary tuberculosis was introduced by French phthisiotherapeutists; this was soon followed by drinking fresh beeves blood and at the abattoirs in Paris the daily attendance soon became very large and the drinking of one ½ litre (or 1 pint) of fresh blood once or twice a day became a common practice among the tuberculous visiting these institutions, taking the supposed cure. It only began to decline and ultimately was abandoned when it became apparent that the treatment, how-

ever faithfully and persistently it was followed, would not affect a cure. Today it is only a memory of one of the many things which have been recommended as a cure for tuberculosis and have fallen by the wayside as of no special value.

(C) **Lactotherapy. Milk Cure.** (62) The milk cure in Pulmonary Tuberculosis. Since time immemorial the use of fresh cow's milk has always been highly recommended for the treatment of pulmonary tuberculosis, and at the present time, cow's milk is the most important single article of diet in the treatment of this disease. While some clinicians have advocated its use in large quantities, others again have recommended it in small amounts, as the latter say not to overtax an already disturbed heart and from giving a few glasses daily to that of a quart or more, varying amounts are used. Recently the use of cow's milk in enormously large quantities in the treatment of pulmonary and the various other forms of tuberculosis has attracted a good deal of attention and from some quarters very favorable results have been reported. It is generally conceded that in milk we have the most nutritious single food known, but some maintain that if given in too large an amount it may do harm, that although we have in milk all the units necessary to sustain life and to build up the body, that the quantity of fluid given in milk is generally too great to be taken care of by the heart of the tuberculous individual. That this contention can not be supported we may conclude from the fact that many a tuberculous individual even in an advanced stage of the disease has taken an exclusive milk diet for months at a time without embarrassing the heart.

We must remember that in taking the milk cure it must be an exclusive milk diet — nothing but milk. Pure, unskimmed cow's milk is usually given, and almost from the very beginning of the treatment the patient drinks large amounts, taking it at regular intervals. Usually the patient begins the treatment at 7 o'clock in the morning, drinking a glass of milk every half hour, continuing this during the day until 9 o'clock in the evening. It is a strictly milk diet, incurs no hardship after the first few days, and the taking of a quart to six or more quarts a day causes no discomfort and patients being known to have taken from two and as high as three gallons a day without any inconvenience. To keep the bowels soluble these patients receive a daily enema of salt water, and to favor the elimination

of such large quantities of water a daily warm bath and rub down is advised. Some patients taking the exclusive milk diet complain of slight pain in the stomach or of distress in the abdomen, of nausea and even vomiting is occasionally complained of. In such cases the eating of an orange, sucking of a lemon or eating some grapefruit, by inhibiting the formation of hard, cheesy curds, often gives prompt relief. The citrate solutions like sodium or potassium citrate have long been in use as drugs which favor the formation of light, floculent, cheesy particles when given with milk; these are more readily digested. In taking the cure, pure full milk is usually recommended; however, occasionally the course may be varied, giving at times skimmed milk, then changing off to buttermilk for a day.

The milk given should not be too rich in butter fat and for that purpose the milk obtained from a Holstein herd will be more suitable than that from the Jersey cow. Holstein milk contains about 3.5% of butter fat against the Jersey 5%. Milk contains all the necessary requirements of a perfect diet. 100 grams of fresh cow's milk contain 87 grams of water, 3.5 fat, 5. sugar, 3. casein, 0.5 albumin, 0.75 salt and .24 milligrams of iron. As the human body requires from 12 to 15 milligrams of iron in the daily diet, in about six quarts of milk this amount of iron is present, and as the body while at rest requires about 3000 calories, with light exercise about 3500, this number of heat units is also found in about 6 quarts of milk, and as every quart of good Holstein milk contains about 600 calories, 6 quarts will just equalize the daily waste and repair of the human body. The exclusive milk diet must continue daily for months, sometimes the regular diet is substituted for a month or so and then a return to the milk diet and so on. The milk diet is said to be very successful in arresting or checking the tuberculous process. Many cases are put on a fast for 24 to 36 hours before beginning the exclusive milk diet. An exclusive milk diet may be very helpful in carefully selected cases, and here as in everything else, in every other treatment of pulmonary tuberculosis, we must individualize.

(5) **Time.** Of the necessary factors to bring about an arrest of the tuberculous process, the fifth is time. As the tuberculous disorder usually comes on slowly and insiduously, so the arrest of the process also becomes slow and long protracted. It is estimated that the average duration of a case of pulmonary tuber-

culosis is about two years and it takes also approximately about that length of time to bring about healing. This long drawn out course often becomes very discouraging to the patient when he observes how long it takes to bring about a cure. I frequently draw a simile, namely, that of moving a mountain of sand with a teaspoon, that after days of toil the mountain does not appear smaller, but just continue in the work and you will observe that the pile of sand which you are taking away steadily grows and so it is with taking the tuberculosis cure, it will take a long, long time to notice any change but keep on and persevere and you will observe that first some improvement and finally an arrest of the tuberculous process has been achieved. The patient from the very onset when he begins taking the cure must be thoroughly informed of this fact, that it is a long drawn out affair; he must be constantly encouraged by the physician in attendance, by the nurses, by attendants and visiting friends and he will soon begin to realize the truth—that time plays a most important role and he gradually becomes reconciled to this fact.

(6) **Obedience.** Discipline. The sixth and last but equally necessary factor to bring about an arrest of the tuberculous process is obedience, that is strict obedience on the part of the patient, a perfect submission, the ignoring of which generally spells a grave prognosis. The tuberculous individual must have full confidence in his medical adviser and follow implicitly the given instructions. The psychology of the tuberculous subject is frequently most peculiar. He wants to get well, is very anxious, but usually wants his own way in getting well. He must from the onset be told that he can get well but that no tuberculous patient ever got well who wanted his own way. The physician should know what is best for the patient and he must follow the given directions to the letter. It is only by such means that a healing of the disease is brought about.

In beginning the treatment in a case of pulmonary tuberculosis in whatever stage, be it incipient or advanced, it becomes the physician's imperative duty to co-ordinate these six necessary factors for bringing about an arrest or possible cure of the disease; however, should he not be successful in co-ordinating all six, his efforts will be futile. He must from the very beginning impress the patient with the importance of these six necessary factors in order to have a hearty co-operation and a perfect harmonizing of all.

Symptomatic Treatment. While the tuberculously diseased individual is being cared for, is taking the cure, is trying to bring about an arrest of his disorder, be it while he is still able to be about and coming to the physician's office or to the dispensary, is still ambulatory, or perhaps he is taking the house, bedfast or the sanatorium treatment, certain definite symptoms will arise and must be combated as they manifest themselves. A symptom which is usually present from the onset of the disease and generally tends to remain up to the close of the scene is:

(1) **Fever.** A temperature ranging daily from 99 to 102° and over; here absolute rest finds its greatest application. If the temperature is 102° and over, sponging with cold water or an alcohol rub may be directed. In some instances the temperature reading when taken by mouth may appear normal or but slightly above, but the palpating hand of the examining physician demonstrates that there is fever; in such instances the rectal temperature reading will be the correct one. I have very frequently observed especially at the bedside when the tuberculous individual has been taking the rest cure for some time that the mouth temperature reading is or approximates the normal but that the body warmth and the rapid pulse attracted my suspicion that the reading was faulty and that when a thermometer was placed into the rectum it registered from 1 to 3 degrees more. I firmly believe that in pulmonary tuberculosis the rectal temperature reading is the only correct one. I have often observed in cases with a rapid pulse and a temperature reading by mouth of 99° that a rectal reading frequently showed 101.5 to 102.5° and more.

If there is much temperature, it is always advisable to give a small amount of calomel, say about 1/10 grain every three hours for a few days. A small amount of calomel may be very conveniently given by combining it with a number of antipyretics, as for instance, 1/10 grain of calomel with 2 grains of quinine given in a capsule or with 2 grains of sodium salicylate or two grains of each with a quarter grain of calomel if there is constipation or if the abdomen feels bloated. As a combatant for fever in the tuberculous Pyramidon is the antipyretic *par excellence,* a 5 grain tablet being given once in three hours. This with cold sponging if the fever is high is usually followed by good results. Of the coal-tar products, if given, some have a

decidedly depressing effect on many tuberculous subjects; hence they should be given with caution. Salol particularly has a tendency to show this in a most pronounced way in individual cases. Aspirin, antipyren or phenacetin may be given in small doses, not to exceed 5 grains, and if not given for too long a period may all be very serviceable. The ordinary fever mixtures so much in use in the treatment of the acute diseases, the liquor ammonii acetatis in teaspoonful doses, citrate of potassium in 10 grain doses, sweet spirits of nitre in 10 minimum doses are all very useful. The following combination makes a most grateful fever mixture:

Potassium Citrate ....3ii (8.0 gm.)
Sweet Spirits of Nitre............................3ii (8.0 cc)
Liquor Ammonii Acetatis sufficient to make 3 ounces..(90.0 cc)

Give a teaspoonful in a mouthful of water, sweetened, every three hours.

Tincture aconite root, tincture gelsemium or tincture veratrum viride most favorable remedies in the high fevers of the exanthemata, are of little value in the treatment of tuberculosis; however, in this the pulse must be the guide. In tuberculosis the pulse is usually of low tension; should, nevertheless, the tension be high and bounding, then they may be administered. If the fever is high and tuberculosis complicated by cardiac disease then the tincture of digitalis in 10 minim doses should be given. Frequently after the meal the temperature rises in the tuberculous; in such cases the giving of antipyretic remedies some hours before the taking of food often checks the rise. Fever usually results from the presence of secondary organisms and their toxins in the body and these with the products of bacillary growth and perhaps some tissue destruction and the absorption of all these toxic products bring about a rise in temperature; for this absolute rest is the ideal remedy. Altitude is one of the surest methods for combating fever. In about 70% of all cases of pulmonary tuberculosis with high fever, the temperature is reduced very quickly while residing up in the mountains and patients with high temperature, in whom the fever is not reduced after a two months' stay in the mountains usually presages a grave prognosis unless there are other causes than tuberculosis which keep up the temperature. The 30% of cases which do not improve while residing in the mountains should be removed to the seashore or into the valleys or at any rate a change should be suggested.

(2) **Pulse.** A rapid or fast pulse ranging anywhere from 100 to 120 or more, a tachycardiac, is generally present with the fever. The pulse is usually rapid, readily compressible, there is a want of force and it is very weak. This often becomes a very distressing symptom which greatly alarms the patient and here sedatives are mostly indicated. The giving of an 1/8 or a 1/4 grain of morphine, hypodermically, gives prompt relief, or codeine in 1/4 to 1/2 grain doses either by mouth or subcutaneously, may be substituted, however, all sedatives should be given most cautiously and only in extreme instances. Sodium bromide in 10 to 20 grain doses once in three hours is often very beneficial. Tinctura opii deodorati, in 5 to 10 drop doses, tinctura hyoscyami in 10 drop doses every three hours with perhaps the addition of 5 drops of the tincture of digitalis may be of good service in slowing the heart's action and the giving of cold drinks and the application of the ice bag over the heart is much in favor in some quarters. Avoid all excitement, inhibit the use of intoxicants in all forms and do not give hot or warm drinks, but give freely of cold water.

(3) **Cough.** This in pulmonary tuberculosis is the most distressing of all symptoms. What to give to relieve the cough often taxes the skill of the attending physician to the utmost. In the very active and progressive cases cough is usually a constant and very annoying symptom whilst in other cases the cough pursues a more quiet course, but we must also remember that in some cases of pulmonary tuberculosis this one symptom of cough may be entirely absent. The cough may be dry or moist, productive or non-productive, and the expectorations be very slight on the one hand and very profuse on the other. The treatment here consists of much rest in bed, and as the air about the room is usually very dry, and this has a tendency to irritate the throat and this increases the cough, the aim should be to keep the air a little moist; this is best accomplished by placing about the room and near the bed a basin of hot water. The giving of morphine in very small doses 1/20 to 1/16 grain or codeine in 1/12 to 1/8 or heroin hydrochlorate in 1/24 grain dose is always indicated. Always be cautious in giving opium or its derivatives. Tincture of hyoscyamus in 10 minum, tincture of cannabis indicae in 5 drop, terpin hydrate, sodium salicylate, aspirin, ammonium chloride or carbonate either separate or combined in 1 or 2 grain doses every 2 or 3 hours have found

much favor. The well known pharmacopeal preparation, the Mistura Glycyrrhizae Composita, compound licorice or brown mixture, given in one or two teaspoonful doses is a most valuable, reliable and dependable cough medicine. This can be somewhat improved by the addition of two to five grains of either ammonium chloride or carbonate to each teaspoonful. The old household remedy known as paregoric, the Tinctura Opii Camphorata of the pharmacopea in one or two teaspoonful doses is equally a reliable remedy. The various combinations of terpin hydrate with codeine or heroin, the different compounds of White Pine Syrup offered for sale, the syrup of tar, of ipecac, of squill are all more or less valuable as preparations for the relief of cough. Some of the cough preparations or mixtures which I am wont to prescribe are the following:

(a) Ammonium Chloride drachm one..................(4.0 gm.)
Syrup of Senega,
Syrup of Ipecac each drachms four................(15. cc)
Mixtura Glycyrrhizae Composita sufficient to make three ounces ....(90.0 cc)

Directions: A teaspoonful with water once in two or three hours.

(b) Ammonium Chloride drachm one..................(4.0 gm.)
Syrup of Senega,
Syrup of Squill each drachms four................(15. cc)
Elixir Terpin Hydrate and Heroin sufficient to make three ounces ....(90.0 cc)

Directions: A teaspoonful every two to four hours in water.

(c) Ammonium Carbonate,
Terpin Hydrate each drachm one..................(4.0 gm.)
Elixir Terpin Hydrate and Codeine,
Compound, Mixtura Glycyrrhizae of each sufficient to make three ounces ....(90.0 cc)

Directions: Give a teaspoonful with water every two or three hours.

(d) Ammonium Carbonate,
Ammonium Chloride each one drachm............(4.0 gm.)
Syrup of Senega,
Syrup of Squill Compound, each three drachms....(12.0 cc)
Syrup of Tolu or
Syrup of Wild Cherry Bark sufficient to make three ounces.. .... (90.0 cc)

Directions: One teaspoonful with a mouthful of water every three hours.

Many other efficient combinations could be enumerated here, but the physician will have to prescribe as each case requires.

In the old chronic forms of cough, not painful or distressing but perhaps copious, I much favor the following:

Take: Creosote (Beechwood).
Guaiacol, pure.
Oil of Cinnamon.
Oil of Cloves, each one half or one drachm (2.0 cc) or (4.0 cc)
Give 2, 3 or 4 drops on loaf sugar every 2, 3 or 4 hours.

Creosote or its derivatives, Guaiacol, Thicol may be given. Avoid as much as possible the giving of narcotics or better give them sparingly. The physician must always bear in mind the nature of the drug which he is using for the allaying of cough, that there is always a strong tendency to give the drugs which enslave, because it is these that seem to give most relief and for which the patient will repeatedly ask; if given at all they must only be used in most minute doses.

(4) **Night Sweats.** A most distressing, annoying and alarming symptom coming on usually about midnight or towards the early morning hours, about 4 o'clock, when tissue relaxation is at its height. This symptom is often caused by the toxins present in the organism. It may also at times be due directly to mechanical causes, such as too much bedding, or clothing, to overheated rooms or insufficient and poor ventilation or again the patient may be tucked in too close in bed, or it may be due to extreme nervous depression. Whatever may be the cause, this should first be ascertained, if possible corrected, and the patient made comfortable. If from nervous causes the administration of a sedative ⅛ grain morphine with perhaps 20 grain sodium bromide is often efficacious, inducing rest and sleep. Often thorough ventilation of the rooms will inhibit the night sweats; however, the most common cause is from the disorder itself, from the elaborated toxins and from the toxins of tissue destruction causing the low tensioned pulse and the low blood pressure, all combine to produce vaso-motor disturbance, capillary relaxation, stasis and sweating. As these patients in addition to the sweating have a slightly accelerated pulse and a temperature, alcohol sponging often gives much relief. In those who are somewhat advanced in years, a teaspoonful of whiskey or brandy given in a little hot water, sweetened to suit the taste, at bed time, will very frequently retard the sweating. Drinking a cupful of hot sage tea prepared by steeping an ounce of sage leaves or sage tea in a pint of hot water and then allowed

to simmer for about five minutes strained and sweetened to suit the taste, will frequently be found very satisfactory.

Many prefer giving the sage tea as a cold infusion, and then in some again a glass of cold milk taken at night will give relief. However, here the use of drugs will find its must suitable application in these symptomatic disturbances if given in appropriate dosage, foremost of which will be found atropine sulphate in 1/200, 1/150 or 1/100 of a grain given either by mouth or more efficiently hypodermically. If much restlessness or nervousness is present the addition of a little Morphine sulphate in 1/16 to 1/8 grain doses is indicated. The aromatic sulphuric acid given in ½ to 1 teaspoonful doses, well diluted with pure cold water, and drank during the evening hours is very often sufficient and useful. Agaracin, a glucoside prepared from a fungus, the Agaricus Albus, given in 1/10 grain doses at bed time will be found a most reliable drug in many cases of excessive night sweats. Camphoric acid in 2 to 5 grain doses by mouth, giving a few doses during the evening hours is with many a most favorite drug. Many use the newer hypnotic drugs like veronal, adalin, sulphonal, trional, etc., in 5 grain doses. Sodium bromide in 20 grain doses dissolved in a mouthful of water and given during the evening is often most helpful. The various preparations of valerian, the tincture, the fluid extract, elixir of ammonium valarianate, etc., may all be employed.

(5) **Dyspnoea. Shortness of Breath.** A very annoying symptom. Here again as in the general treatment of tuberculosis, rest is the most effective and prompt therapeutic remedy. At the very onset we must try and ascertain the cause of this shortness of breath. It is often due to an accompanying cardiac disorder or perhaps a kidney complication, or it is simply because the pulmonary parenchyma is greatly destroyed or contracted, is perhaps distended, emphysematous or atelectatic, all conditions which greatly interfere with the free access of air to the alveoli. If the cause can be ascertained, the proper remedy may often be very promptly applied; however, no matter what may be the cause of the dyspnoea, a small dose of morphine is usually the drug which gives prompt relief. Should much cough accompany the dyspnoea, the addition of a small amount of atropine sulphate 1/200 or 1/150 grain is very useful, or a hypodermic tablet may be given or administered by mouth. If much nervousness accompanies this distressing symptom, Sodium Bromide in 15 or 20

grain doses is indicated. The following is a most admirable combination:

Sodium Bromide drachms four ....(15.0 gm.)
Morphine Sulphate grain one ....(0.06 gm.)
Essence of Pepsin sufficient to make two ounces.......(60.0 cc)

Directions: Give one teaspoonful in a little water at once, repeat in 2 or 3 hours if necessary.

If the shortness of breath is due to an accompanying cardiac disturbance, then the addition of tincture of digitalis in 5 to 10 drop doses added to the ordinary mixtures is advisable. If the heart's action is feeble, pulse scarcely palpable, with little force, then the use of strychnine in 1/60 to 1/40 grain doses hypodermatically is often most useful. Aromatic spirits of ammonia in ½ to 1 teaspoonful doses given in a few mouthfuls of water, or a little whiskey or brandy, is often found to be a most helpful remedy, and alcohol sponging especially if there is fever will often be followed by better breathing conditions. A most souverain remedy, one of much value for the relief of shortness of breath is oxygen gas. This gas should be inhaled through the nostrils by means of a rubber tube tipped with glass tubing. If the dyspnoea is not very urgent this gas may be given interruptedly, but not if the shortness of breath is lasting; it should then be given continuously for hours, even for days, and the patient should be asked to inhale deeply in order to force the oxygen gas into the very recesses of the lungs so as to become more readily effective.

(6) **Sleeplessness.** Another very annoying, even painful symptom. Here again the anodynes and sedatives take the leading role, of which may first be mentioned morphine sulphate given hypodermically in 1/12 to 1/4 grain dose according to the exigency of the case. Sodium bromide in 10 to 20 grain doses dissolved in a few tablespoonfuls of water given every two or three hours for two or three doses, beginning the administration in the middle of the afternoon, will often induce refreshing sleep. Trional, sulphonal or veronal in 5 grain tablets once in three hours for a few doses, Dover's Powder in 10 grain doses, Tinctura opii deodorati in 5 to 10 minim doses, camphorated tincture of opium or ordinary paregoric in one or two teaspoonful doses given in cold water are all more or less useful and helpful. In elderly individuals a teaspoonful of whiskey or brandy given

in a little hot water and sweetened to suit the taste is often followed by restful sleep.

(7) **Gastro-Intestinal Disturbances in the tuberculous are often** very distressing. Nausea, vomiting, dyspepsia, constipation, etc. The use of ice, by mouth, in small pieces will often allay the sickening stomach symptoms of nausea or vomiting. The giving of essence of Pepsin in ½ teaspoonful doses every ¼ to ½ hour without water will be found very serviceable, and if much distress from gaseous eructations after eating then the various combinations of sodium bicarbonate will be found beneficial.

Sodium Bicarbonate ounce one ....(30.0 gm.)
Magnesium Calcined ounces two ....(60.0 gm.)
Oil of Peppermint drops three........................(0.2 cc)

Mix well. Give one half to one teaspoonful in ½ glass water, repeat in ¼ or ½ hour if necessary.

Many tuberculously diseased individuals are also suffering from a hypoacidity and consequently after the ingestion of a meal, particularly if rich in protein, have much gastric distress. Here the giving of a small amount of diluted hydrochloric acid often gives relief. The following can be offered as a reliable combination:

Acid Nitro-muriatic, diluted
Tincture of Nux Vomica, each drachms two............(8.0 cc)
Tincture of Gentian Compound ounce one..............(30.0 cc)
Glycerine sufficient to make three ounces..............(90.0 cc)

Directions: Give a teaspoonful in ½ glass of water ½ hour after meals.

For constipation, the aromatic fluid extract of Cascara Sagrada in teaspoonful doses in ½ glass of water at bed time or a pill of Aloin, Strychnine, Belladonna and Ipecac given alone or with a 1/20 or 1/10 grain calomel is often very efficient. Although constipation is not a frequent symptom accompanying tuberculous disease, if present it is always better to regulate it by an appropriate diet. To accomplish this a liberal supply of vegetables like spinach, cabbage, celery, lettuce, etc., or what is known as rough food should constitute a large part of the principal daily meal. The addition of a few tablespoonfuls of ground linseed or flax seed meal or bran to the ordinary breakfast foods will be found most serviceable in all constipated conditions in the tuberculous as well as the non-tuberculous individual. The newer preparations, the liquid petrolatum extensively recommended for

the cure of constipation, are also very valuable and reliable in some individual cases, in appropriate doses and salt water enemas are freely recommended if applied by a trained person. Avoid above all the giving of active cathartics.

(8) **Diarrhoea.** This symptom is frequently present when tuberculous peritonitis or enteritis accompanies the pulmonary disorder. It is fully described in Chapter 29—Tuberculous Peritonitis, page 388.

(9) **Pleurisy Pain.** For the relief of pleurisy pain. See Tuberculosis and Pleurisy, page 335.

(10) **Tachycardiac.** See Tuberculosis and the Cardio-vascular System, page 450.

(11) **Urinary Distress.** See Tuberculosis of the Genito-Urinary Organs, page 410.

(12) **Abdominal Pain.** See Tuberculous Peritonitis, page 388.

(13) **Sore Throat.** See Tuberculous Laryngitis, page 369.

(14) **Hemorrhage.** See Tuberculosis and Pulmonary Hemorrhage, page 362.

Note. Tuberculin as a therapeutic remedy in the treatment of pulmonary tuberculosis it is now somewhat obsolete, however, in some quarters it is still used and as stated with very satisfactory results. The opinion of many phthisotherapeutists is that what effect for the better in the use of tuberculin may be observable in pulmonary tuberculosis must be attributed chiefly to a psychologic influence *per se,* and in no way to the drug in question. The use of tuberculin in the treatment of pulmonary tuberculosis may be found in Chapter XXIV—Tuberculin: Indications and Contraindications, page 316, to which the reader is referred.

CHAPTER 20

THE CARE OF THE TUBERCULOUS—(Continued)

The Surgical Treatment of Pulmonary Tuberculosis (33)

Introduction. Under surgical treatment of pulmonary tuberculosis should be considered all those methods of procedure where by means of surgical measures we aim to give the tuberculous individual relief, either from pain or suffering or from excessive fluid in the chest cavity, if by its presence the lung becomes crowded and breathing is strikingly interfered with; for the relief of urgent dyspnoea; for the relief of uncontrollable hemorrhages, but perhaps most frequently for the purpose of bringing about, if possible, an arrest or cure of a pulmonary tuberculous process and this chiefly by putting the lungs at rest.

The simplest method, and the one now in general use, for bringing about an immobilization of the lungs for the treatment of pulmonary tuberculous disease is by means of artificial pneumothorax. If from extensive pleural adhesions this procedure is not possible, then the other, the more difficult and more serious thoracoplastic operation may be advocated. It seems that the effect of lung compression is a lowering or lessening of the circulatory lymph flow through the collapsed lung, a lessening of the amount of oxygen and an increase in the CO_2 content, all of which is unfavorable to bacillary growth and the spreading of the bacilli through the blood or lymph stream, inhibiting the flooding of the organism with toxins.

The various operative methods in vogue in the treatment of pulmonary tuberculosis, namely, artificial pneumothorax, pneumonectomy, phrenicotomy, thoracoplasty, chondrotomy, etc., when compared with the dietetic-hygienic and medicinal will always be considered secondary and the suggestion made that all pulmonary cases be treated surgically can not be applied in practice.

Artificial Pneumothorax or Compression of the Lung. (61) **Historical Notes.** Early in the study of pulmonary tuberculous disease it was noticed by close observers that many cases of

tuberculosis showed a healing tendency if the lung was compressed by mechanical ways, either by fluid or by air; that many cases if accompanied by a severe pleurisy or if the pleural cavity contained both fluid and air and that if this was maintained for some time a healing of the tuberculous process usually resulted. Baglivi as early as 1669 reported his observations on the frequent healing of pulmonary tuberculosis after injuries to the chest wall and opening of the pleural cavity, and William Hewson, 1739-74, made the observation that air is contained in the pleural cavity in pneumothorax. He was one of the first to perform the operation of paracentesis, and Laënnec described and differentiated spontaneous pneumothorax from the other pulmonary disorders. It became apparent from these observations that if nature effected a healing by putting the lung at rest, an artificial compression of the lung may be equally as efficient. Although the signs and symptoms of a spontaneous compression of the lungs had been known since the time of Hippocrates, nothing definite is recorded until the beginning of the 18th century when Jean Marie Gaspard Itard, a French surgeon (born at Oraisio in 1775, died in Paris in 1838), wrote a treatise on pneumothorax. He was the first to investigate the causes of natural pneumothorax and he has given us a clear description of a number of cases in which both air and water were present in the thorax during the course of tuberculous disease. About the same time James Carson* read before the London Medical Society (namely on November 25, 1819) a paper "On the elasticity of the lungs."

He was the first to study and to note the contractive power and elastic quality of the lung. Both Itard and Carson make mention of the fact, although this had been noted before their time, that injuries to the chest wall like stab wounds about the thorax of tuberculous soldiers were often followed by a traumatic pneumothorax which generally would show a marked healing tendency and even often effected a cure of the pulmonary tuberculous process. Carson's observation led him to intensive work and we find that in 1822 he published his first papers giving the results of puncturing the chest wall in rabbits. Although his technic from present knowledge of paracentesis may be considered somewhat crude, still he gives us a very clear picture of the behavior of these animals during the ordeal. Making an incision

*(Essays, Physiological and Practical by James Carson, M. D., physician in Liverpool, appeared in 1822.)

between the ribs he notes that the animals remained for a few seconds as if stunned but that at the end of a few hours they would resume their usual normal state. He concluded from this that collapse of one lung may be done with perfect safety on the human and suggested the possibility of treating pulmonary hemorrhage by lung compression, that it would be equally as efficacious in controlling bleeding of the lung as contraction of uterus would be in uterine hemorrhage. It is stated, supposedly authoritatively, that Carson assisted by Bickersteth actually treated pulmonary tuberculosis by artificial compression. Ramadge in 1836, advocated puncture and compression of the pleural cavity as the best remedy for the treatment of pulmonary tuberculosis and reported that his patient was doing well at the end of two years following the operation. Houghton, a co-worker with Ramadge, states: "The constant movement of the lungs, the entering air during the respiratory act keeps up a continuous stimulation and an increase of blood to the tuberculous tissue, this favors growth and extension of the tuberculous process. In immobilization of the lung this ceases nearly altogether because the organ is put to complete rest and now lies in a more or less anemic condition close to the spine." Luigi Parola, physician at the hospital at Cuneo, Italy, published in 1849 a 700 page volume on pulmonary tuberculosis in which he mentions the work of Carson and of Barry of one hundred years ago and refers to this treatment. Constatt, 1843; Wunderlich, 1856; Ehler, 1867; all mention Carson's work and they all mention his animal experimentation but we glean nowhere that artificial compression of the lungs was actually performed by him on man. Although he suggested the procedure in 1821 and it was again advocated by Spaeth, a German physician in 1870, no artificial lung compression was attempted on the living human until the time of Forlanini of Padua who first put this method of treatment into actual practice, reporting his observations first in 1882 and after years of close labor publishing his results at the International Tuberculosis Congress at Rome in 1894. In America up to the close of the last century nothing was done along these lines with the exception of the work done by J. B. Murphy (67) and his associate, A. F. Lemke (66) of Chicago, the former reporting their observations in 1898 and the latter his in 1899. J. B. Murphy in his oration, "Surgery of the Lung," delivered at the 49th annual meeting of the American Medical

Association at Denver, Colorado, June 1898, described the method of procedure in detail and reported five cases in two of which they were not successful owing to pleural adhesions. The time of observation after compressing of the three successful cases was only two and one-half months before the reporting—too short a time to be of much clinical importance. A. F. Lemke, associated in the work with J. B. Murphy, reported the following year his observations on fifty-three cases of pulmonary tuberculosis, giving his findings in a paper read at the Fiftieth Annual Meeting of the American Medical Association held at Columbus, Ohio, June 1899, entitled "Pulmonary tuberculosis treated with intrapleural injection of nitrogen with a consideration of the pathology of compression of the tuberculous lung." His observations were on fifty-three patients from September, 1898. to April, 1899, covering a period of about eight months. He also encountered in some of his cases a thickened pleura, consequently was unable to pass the gas or collapse the lung.

Owing to the untimely death of Dr. Lemke (he being himself a victim of the white plague soon after the reading of his paper) nothing more was heard in American Medical Society meetings about pneumothorax until about 1911 when the subject was again brought forth as a curative measure in pulmonary tuberculosis.

During the earlier years of this century much intensive work on lung compression was done in Europe, and in 1906 Prof. Ludolph Brauer, Marburg, published his report of the most brilliant experimental and clinical work along these lines, and in 1908 Dr. Fr. v. Sauerbruch,(147) Marburg, "Surgical treatment of pulmonary tuberculosis"—Page 82, Vol. 11, Sec. 111; Dr. Hermann v. Schroetter, (140) Vienna,—"Recognition of pure pneumothorax without exudate in pulmonary tuberculosis"—Page 1165, Vol. 1, Sec. 11; Dr. Lucius Spengler,(152) Davos, "The course of the tuberculous disease under the influence of artificial pneumothorax,"—Page 1169, Vol. 1, Sec. 11; presented most interesting clinical observations on artificial collapse of the lung, the reports of which were read and well received at the Sixth International Tuberculosis Congress, Washington, D. C., 1908. At this meeting no other papers on pneumothorax in the treatment of pulmonary tuberculosis were read or offered by either American or other European clinicians. The real and permanent interest taken in artificial pneumothorax by clinicians

and surgeons of our country dates back to 1912 when Louis Hamman and Martin T. Sloan,(55) Baltimore, and Mary E. Lapham, Highland, N. C., presented their observation on collapse of the lung before the annual meeting of the Association for the Study and Prevention of Tuberculosis at Washington, D. C., the latter reporting her observations on thirty-one cases of pulmonary tuberculosis treated by compression of the lung, the former fifteen cases divided into four groups. These papers, the pioneer papers on pneumothorax by American physicians, excepting the papers by Murphy and Lemke, did not receive sufficient encouragement in the discussion and the paper by Mary E. Lapham which was also read at the meeting of the A. M. A., at Atlantic City a short time after was even severely criticised as being of little importance and of doubtful value. It was not until two years after when Gerald Webb of Colorado Springs,(55) Colorado, read a paper at the meeting of the National Tuberculosis Association that phthisotherapeutists became thoroughly aroused to the importance of this valuable therapeutic measure; fourteen leading clinicians participated in a lively discussion and since that time up to the present at every meeting of the National Tuberculosis Associations one or more papers were presented.

These were the first papers presented on the subject of pneumothorax since the organization of the National Association for the Study and Prevention of Tuberculosis in 1904. In the transactions of the Association issued annually since the first meeting at Washington, D. C., in the spring of 1905 until the gathering in 1912, mention is nowhere made of any observation on this method of treatment.

It appears that Rothschild (170) (San Francisco, Cal.) in 1911 reported eighteen cases of pulmonary tuberculosis treated by lung compression and Robinson and Floyd in the Archives for Internal Medicine in April, 1912, described artificial pneumothorax more in detail.

Resumé from historical data. The idea of compression of the lung for the probable arrest of the tuberculous process originated with Carson, although his suggestion applied to what is known as open pneumothorax in distinction to what is now practiced as closed lung compression, but to Forlanini belongs the credit and the original idea of a pneumothoraxtherapy under aseptic conditions, rather than to Carson who suggested simple or open pneumothorax. Following Carson comes Piorry who suggested

a compression of the chest wall for the treatment of pulmonary disease by means of a tight binder or adhesive plaster. By these external means he aimed to bring the walls of cavities into close apposition and he even applied heavy weights, as much as 14 pounds, securely fastened over circumscribed spots, to compress the thorax so as to immobilize the diseased and not the healthy portion of the lung, that this might have a tendency to bring about scar tissue formation. The idea of Carson and the suggestion of Piorry were both rejected by the profession, at the time, as being entirely impractical. No definite data as to Carson's work on produced lung compression on the human are obtainable and for more than a half century the method was nearly entirely forgotten until reintroduced by Prof. Carlo Forlanini, University of Pavia (Padua) who in 1882 again called attention to lung collapse by closed pneumothoráx, reporting in 1894 his observation to the International Tuberculosis Conference at Rome on the healing and cure of pulmonary tuberculosis by means of artificial pneumothorax. Between the year 1821, that is, from the time of Carson until 1882 when Forlanini rediscovered immobilization of the lung for the arrest of a tuberculous process very little work was done and that only in isolated instances. Forlanini's observations and study form the basis of the present applied method for the arrest of pulmonary tuberculosis by means of an induced pneumothorax. He pointed out that a complete inhibition of functioning is necessary, that there must be an absolutely complete uninterrupted and continuous immobilization and that a simple lessening of pulmonary motility favors an extension of the process. As to priority, we may state the words of Forlanini: "It is not a matter of priority,—that belongs to Carson. No doubt that Carson in 1821, I (Forlanini) in 1882 and Murphy in 1898 had the same identical thought or idea and that it may be said there are indeed three priorities, that the first belongs without doubt to Carson, that his suggestion was lost for more than a half century and was then rediscovered by me (Forlanini) who may be credited with the second priority, and that the third priority is due to Murphy assuming that he had no knowledge of my (Forlanini) work." Forlanini's (the latter's) paper was read in 1894 before the International Tuberculosis Conference in Rome and appeared right after in full detail in the Müenchner Mediciniche Wochnschrift. See spirited controversy as to priority. Forlanini-

Padua, Daus—Gentrogotz, Berlin. Die Therapie der Gegenwart 1910.

Observations on compression of the lung by means of gas, air or by fluids; serous, seropurulent or purulent. The respiratory act and the bony framework of the chest offer a constant resistance against pulmonary cicatrization and rest. This resistance to nature's efforts at repair exists about every pathologic pulmonary cavity. If, however, we can compel the abscess cavity to collapse and to empty its contents, it will heal; an observed physiologic phenomenon. If an abscess cavity can be obliterated and subsequently healed by forcing it to collapse, might not pulmonary tuberculous processes be arrested on similar lines by mechanical immobilization, collapse and enforced rest? This was the argument advanced by those intensely interested in the pulmonary tuberculosis problem. This led up to the induction of the artificial collapse of the lung so much now in favor. Quoting from Murphy's paper, "Have we any evidence from postmortem records that will support the principles of lung immobilization and physiologic rest, and which would favor the healing of the pulmonary process? Yes we have, namely, (1) lungs that have been severely involved in a tuberculous process when repaired show a contraction of the overlying chest wall in proportion to the degree of tissue involved; (2) tuberculous lungs compressed by pleural exudates generally heal during the collapse if the compression is maintained for a sufficient length of time, and, (3) lungs allowed to be collapsed following an operation for empyema have usually healed during the resting process.*

*Attention is directed here to the clinical observation that more than 80% of empyemas in the adult are secondary to a primary pulmonary tuberculous involvement.

Every general practitioner can recall cases of tuberculosis of the lung resulting in empyema[1] or hydrothorax in which there was complete recovery of the tuberculous process when the empyema or hydrothorax was operated at the proper time. This may not apply to those cases of advanced pulmonary tuberculosis in which much tissue has been destroyed and in which much cavity formation or consolidation and fibrosis has taken place,

[1]The most probable reason why empyema is more curative in pulmonary tuberculosis is the fact that the pus is never absorbed but may increase and continue the lung compression more evenly, whilst in pleurisy with effusion there is always a tendency at absorption and consequently a relaxation from the compression.

so that the resultant pleurisy or empyema is not capable of producing sufficient contraction or collapse of the pulmonary structure. The number of cases of operated pleurisies or empyemas which present cures of the primary tuberculous process of the lungs carry conviction that compression and collapse of the lung favor a reparative process."

Pulmonary collapse or quiescence may be obtained in three ways: (1) by collapse of the lung by intrapleural injection of nitrogen gas or air. This is generally known as intrapleural pneumothorax or simply artificial pneumothorax; (2) by forcing the collapse of the lung by separation of the parietal pleura and by intrathoracic compression. This is known as extrapleural pneumothorax, and (3) by removing the bony resistance, the ribs, and allowing the collapse of the chest wall, thus favoring contraction and cicatrization. This is known as Thoracoplasty.

To insure a rapid contraction of the lung to favor early cicatrization and to put the lung at rest irritants have at various times been used. Iodine, in the form of the tincture, solution of iodide of potassium, and of carbolic acid, mercuric and other chlorides and many others have been tried with more or less satisfactory results. These solutions were directly injected into the diseased pulmonary tissue anticipating that by their presence sufficient irritation would be brought about to ultimately favor cicatrization. By some surgeons the use of the actual cautery has been advocated. Most all these therapeutic measures have been abandoned for the much simpler, less painful, and more natural method of putting the lungs to rest by means of nitrogen gas or filtered air.

Compression of the lung obstructs mechanically the various avenues through which the infection is transmitted to other parts, the most important of which are the air vesicles, the vesicular ducts, and the smaller bronchi as well as the lymphatic circulation which plays a great part in the spreading of the disease.

That compression of the lung aids the natural forces which are constantly at work in the effort to arrest the tuberculous process we see frequently, as when the disease is complicated by either pleurisy, empyema or a natural pneumo-thorax, and also in the permanent retraction of the chest wall often observed in old and long quiescent or arrested pulmonary processes. The effect of a natural immobilization of the lungs had been observed by clinicians for centuries and even the ancients noted the beneficial result, but the practical application of these observations had not been attempted, and only within very recent years have the principles

observed been applied by clinicians in the treatment of pulmonary tuberculosis.

A spontaneous pneumothorax developing in the course of pulmonary tuberculosis seems to arrest or check the progress of the disease. Some tissues resist completely a tuberculous invasion as, for instance, the fibrous tissue of tendons and fascia; others when attacked never tend to repair, such as the tuberculous ulcer in the intestine, and again others, such as the peritoneum offer great reparative powers or resistance to the advancement or spread of the disease. A comparison of the postmortem evidences of repair of tuberculosis in the various tissues shows that the lungs far exceed any other tissue in the body in its ability to overcome the effects of a tuberculous inoculation.

From the beginning of the tuberculous nodule, the solitary tubercle in the lung which is the first evidence of the body's defense activities, down to active pulmonary tuberculosis there is a constant attempt at histologic repair to check the advance of the disease and to put the diseased parts at rest. A spontaneous pneumothorax in the course of a tuberculous process is usually most effective in producing this, an empyema as a concomitant symptom in tuberculosis has a similar effect and in a less degree a pleurisy with effusion. If we observe carefully we will notice nature's effort at rest in most every case of pulmonary tuberculosis. About the first thing we observe by inspection is a lessening of motion on the affected side to spare the lung, an inhibition of the excursion over the diseased side; the muscles are rigid over the involved areas and there is a contraction or more properly the retraction of the diseased side, all evidence that nature is endeavoring to put the injured parts at rest till she has completed the reparative work. We notice the same in tuberculosis of all the other organs and tissues of the body.

Cases of pulmonary tuberculosis suitable for lung compression. The indications and contraindications. Selection of cases.

It has long been an undecided, and judging from the literature on the subject, still an open question as to what really does constitute a suitable case for immobilization of the lung. In the selection of cases from the incipient to the far advanced and from the fibroid to the hemorrhagic there exists a great difference of opinion. It was early recognized as an established fact in tuberculosis treatment, dietetic, hygienic and otherwise, that the earlier the treatment is begun the more favorable the prognosis and this undoubtedly also applies to the surgical treatment of the disease. In the beginning, advocating compression of the lung for the cure of the diseased organ, the selection of cases was confined chiefly to those in which the other or opposite lung was in apparent perfect health. To show how cautiously then this selection was made we may mention that an experienced operator in selecting his first cases for collapse carefully studied the case

histories of ninety-six patients in the male receiving wards of a large tuberculosis sanatorium and found only seventeen individuals in which the disease appeared to be confined to one side only. From that number two were selected for compression. All others were considered not suitable. With our present knowledge a goodly number of the others perhaps would have been urged to take the treatment and today the consensus of opinion is that many cases of pulmonary tuberculosis are suitable for collapse providing the involvement is not too extensive, or if extensive, confined mainly to one lung; that the tuberculous process is not running a very rapid course; and that the patient is not already in a moribund condition. In short, it may be stated as a rule that all cases of pulmonary tuberculosis that are not terminal, or that are not too active and that when under observation for some time show no improvement in the general condition, seem unable to be arrested, where the pulse remains fast and temperature about 101-102 and higher, and perhaps cases which under long observation seem to show no further progression nor retrogression, may be suitable for attempted lung compression. If, after months of conservative and careful treatment, hygienic, dietetic, medicinal, tuberculin, etc., no satisfactory progress can be recorded, then compression or collapse of the lung should be attempted.

Early in the study it became evident that the most favorable and typical cases for compression were the unilateral, although pure unilateral pulmonary tuberculosis is a rarity, and that next are those bilateral cases in which the involvement is not too great on the opposite side. If there is much evidence on physical examination that both lungs are involved, usually the lung selected for compression is the one in which the disease appears to be the more active. In bilateral pulmonary tuberculosis, particularly in the somewhat advanced cases, attempts at compression of both lungs have been made by first collapsing fully the most involved or active and later by partial immobilization of the opposite lung. This method in the hands of competent surgeons has given most gratifying results. By this procedure the mediastinum is more completely put to rest, inhibiting in a measure mediastinal motion and enabling the remaining healthy lung to perform more quiet breathing.

The most favorable application of lung collapse offering the most gratifying results is in recent and uncontrollable pulmo-

nary hemorrhage accompanying tuberculous disease. In contemplating a compression of the lung for the control of pulmonary hemorrhage the surgeon must be sure from careful physical examintion from which lung the blood comes, so as not to collapse the freely functioning or healthy one, and incidently must be sure that it is a pulmonary and not a gastric, oesophageal, buccal or nasal hemorrhage. This compression for the arrest of pulmonary hemorrhage may best be done by slow collapse during the interval of bleeding to assure more lasting results. However, if the hemorrhage is very severe and profuse, more rapid and quick compression is desired. The question has often been asked, what effect compression of one lung has on the opposite or non-compressed lung which is generally already and slightly involved? If the functioning lung is slightly diseased compression of the opposite lung or putting it at rest will not overtax this lung. Most likely the increased functioning of this lung will increase the blood and lymph flow, and congestion or hyperemia will favor rather a healing of the pulmonary lesion than an extension.

To summarize: Pneumothorax indications and contraindications. Indications: (1) In all chronic destructive processes limited to one lobe; (2) in very acute exudative and infiltrative cases; (3) chronic infiltrative processes with tissue destruction showing a progressive character and which are not influenced by the generally applied therapeutic methods; (4) owing to the unfavorable prognosis basal tuberculosis complicated by bronchiectasis may be considered suitable in some selected cases; (5) as an indicatio vitalis in frequent profuse pulmonary hemorrhages. Contraindications: Basal tuberculosis generally when both sides are extensively involved, tuberculous peritonitis and enteritis, complicating heart and kidney disease, non-tuberculous affections, abscess, gangrene, foetid bronchitis, bronchiectasis, etc., are all unsuitable for lung compression. The contraindications then for the induction of artificial pneumothorax are in terminal cases, in the miliary form, in chronic contracted fibroid phthisis, in pulmonary cases accompanied by serious heart and kidney disorder and in tuberculous enteritis and peritonitis.

A sufficient period of trial without improvement should be the basis or the failure of progressive cases of pulmonary tuberculosis to respond to the ordinary methods of treatment, regardless of the stage, before com-

pression is considered. Numerous factors will determine the decision, such as a rapid extension of the process, marked constitutional disturbances, rapid impairment of the general conditions, etc.

It is the gradually formulating consensus of opinion among the majority of observers that the best results can be obtained before the occurence of extensive adhesions, marked involvement of the opposite lung and the irreparable deterioration of the general resistance.

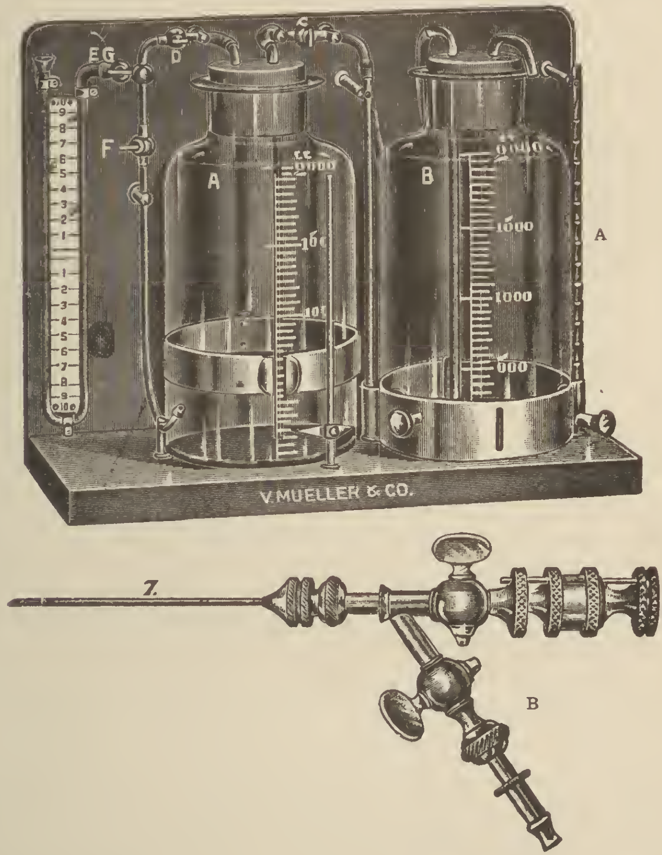


Fig. 39. (a) The Floyd-Robinson Apparatus for Compression of the Lungs; (b) The Floyd-Robinson Needle.

The Apparatus and the Technic for the Induction of Lung Compression.

The induction of artificial pneumothorax or lung compression to put the diseased organ to rest requires usually only a very simple apparatus. The apparatus originally used in this country, devised by the late Dr. J. B. Murphy of Chicago, answered all

requirements. This apparatus provides means for noting the escaping gas, measuring its volume in cubic inches and using pure nitrogen gas for the compression. At the present time nitrogen is but little employed, having been superseded by the use of sterile and filtered atmospheric air and the volume of air used for compression is now measured in units of ccm. There are now many different makes of apparatus to be had. The Floyd-Robinson (98) outfit originally described in the journal of the A. M.A., in March 21, 1914, will answer all purposes. Every apparatus offered is simply a modification of the Murphy-Brauer-Forlanini type to which a water monometre has been attached, as had been suggested by Prof. Chr. Saugmann of Christiania in 1907. This is by all means the most important addition, for it registers with exactness the location of the end of the needle and by its use the amount and flow of the entering gas or air can be regulated with perfect accuracy. A good and complete apparatus may be purchased from any dealer in surgical instruments. As already stated the original compression (99) was done with pure nitrogen gas. This was generally supplied by dealers, delivering it in steel tanks, but now most operators are using pure and filtered atmospheric air which is generally prepared either before or while the operation is proceeding by the following method. A long rubber tube is connected at one end with an intake valve of an ordinary aspirating pump and at the other with a metallic bathroom spray loosely filled with sterile cotton. This end is placed in the outside air which is then pumped through a bottle of sterile cotton, thence into one filled with a 1-1000 bichloride of mercury solution and from this into the inverted cylinder surrounded by boiling water. Another and simpler device for the preparing of the atmospheric air for use in lung compression is by means of two bottles, a few glass tubes, rubber tubing and an ordinary atomizer bulb or pump forcing the air through a solution of carbolic acid and then through sterile absorbent cotton.

The technic for the induction of Pneumothorax is as follows: (63). The necessary apparatus is placed on a solid, well-supported table. The operator and his assistants must be surgically clean. To conserve time one nurse remains sterile while another gets the patient into the operating room and takes care of other details. An assistant or nurse operates the apparatus, reads and records the pressure, the site of injection, incidents,

etc. As a certain number of cases of serous effusion often complicate this treatment, strict observation of the principles of surgical asepsis is imperative. The needles used are 18 gauge aspirating, the ends of which are filed to an angle of 45 degrees and about 1/16 of an inch from the end a nick is filed. These needles are carefully cleansed after use by running cold water and a stiletto put through them several times in order to remove any coagulated blood or tissue. They are then dried by compressed air, stilettos replaced, wrapped in gauze, then covered with muslin and sterilized under high pressure for an hour. A number of needles must be kept on hand to eliminate sterilization during the procedure. A hypodermic syringe filled with camphorated oil should be a part of the equipment. All treatments, initial as well as refills, must be preceded by a thorough physical examination. The patient is stripped to the waist, placed on the operating table lying with the side to be operated uppermost. A sheet is placed over the body below the waist line, then the sides of the chest are thoroughly cleansed from the sternum to the posterior axillary line and from the 4th to the 7th interspace and covered over with two sterile towels. The operator usually selects the site for puncture by means of percussion and his previous observation. The site is usually in the 5th or 6th interspace about the anterior axillary line or perhaps slightly farther back, particularly when the left side is to be injected. The site is then painted over with tincture of iodine and the operator introduces a hypodermic needle so as to make a bleb with a small amount of 0.5 per cent solution of novocaine. After a few seconds the needle is slowly reintroduced perpendicularly and Schleich's infiltration method employed. Care is necessary in order not to puncture the parietal layer of the pleura. The next step is to pick up the integument surrounding the site between the thumb and forefinger of the left hand with considerable pressure, and with a cataract knife puncture the layers of the skin; frequently no hemorrhage follows the pressure method. The index finger is then placed over the interspace so that its tip almost covers the incision. The needle is next held perpendicularly between the tips of the fingers and thumb of the right hand, the wrist resting upon the patient's chest to steady the movement. It is then slowly introduced, the index finger on the wound acting as a guide and the nail as a brake. Occasionally slight negative oscillations are observed in the monometer when

the point of the needle reaches the parietal layer of the pleura, but as soon as the needle passes through this layer into the free pleural space the suction, or negative pressure, immediately causes[2] oscillations varying between 10 or more to 1 or 2 cm Such oscillations of water, even with much less amplitude, give the assurance that the end of the needle is in the free pleural space and that now air may be allowed to flow into it without danger. It is best to wait a few moments in order that the oscillations may become stable before they are recorded and air is admitted. In working without a monometer, and this is extremely infrequent at the present time, to assure oneself that the end of the needle is in the pleural cavity, the patient is instructed to take a few deep inspirations and if the needle is within the pleural space a current of air will be heard rushing in. During the initial operation air should be given at short intervals and no more than 100 cc at a time, the column of water being watched continuously. When the total number of cubic centimetres has been given this is recorded along with the readings following. After the needle is withdrawn the puncture is immediately touched with tincture of iodine and pressure applied, the wound dressed with sterile gauze and the patient instructed to continue pressure for a few moments and, by having the patient then change his position after the needle is removed, the complication of surgical emphysema is very infrequently encountered. A few words of advice as to the use of the compression apparatus. The most vital part is the monometer. It is to the operator what the compass is to the mariner and a disregard for its danger signals will surely spell disaster. In addition to a sustained negative pressure with free oscillations, this the only condition under which the initial insufflation is warranted, there are other readings of great importance. For instance, fluctuations equidistant above and below zero indicate that the end of the needle is in the lung, or, the column of water is uninfluenced when the end of the needle is extra pleural, or is surrounded by dense adhesions or occluded by tissue or blood. A partial occlusion acts as a valve and the readings must be interpreted accordingly. When the water rises slowly on the negative side of

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[2]The intrapulmonary, also described as ordinary atmospheric pressure is usually 6 m.m. higher than the intrapleural. This difference is designated the "negative" pressure. The admission of air into the pleural cavity equalizes this pressure so that it becomes equal to that in the pulmonary tissue. This difference, that is, the pressure in the lungs or ordinary atmospheric and that in the pleural space is registered with the monometer the moment the needle enters the pleural space.

the monometer and the pressure decreases slowly without any oscillations during ordinary respiratory movements, the needle is practically plugged. It should be removed and another needle introduced in the same tract. Failure to locate the free space is often due to coagulated blood and no attempt should be made to reintroduce the needle as long as there is oozing at the puncture. After some compression is produced, the dosage and the necessary length of intervals between refills can be roughly determined by the monometric readings. Some idea of the elasticity of the mediastinum may also be obtained, but not until the pressure approaches zero or becomes positive. The monometer may confirm the diagnosis of spontaneous pneumothorax.

As to the amount of air required for the initial and the subsequent insufflations a wide difference of opinion exists among various operators and whereas one operator limits his first inflow of air to 100 ccm another uses from 800 to 1000 and more. However, practice has proven that it is not necessary to use large amounts—from 100 to 300 at most is usually sufficient for the beginning amount and after this from 300 to 400 may be injected every second or third day until a partial compression becomes established, after which the compression may be repeated once in four to five days until at the end of about six weeks it will be sufficiently collapsed, after which it is repeated about once a week to maintain the compression. As absorption of the retained air takes place more or less rapidly, more so at the beginning of the treatment and less so as time goes on, a refilling about once a month will usually be sufficient. If fluid should follow the injection of air or gas into the pleural cavity, the time for reinsufflation may be postponed. In this manner the treatment may be carried along for months and years.—E. M.

Complications incident to the production of (55) artificial pneumothorax. With the gradual perfection of the technic and a more careful selection of cases, accidents in the course of lung compression are gradually diminishing in number. Whatever is used, nitrogen gas or sterile atmospheric air, it must never be permitted to enter until the monometer establishes the connection with the pleural cavity—by sustained negative pressure and free oscillations. Another important factor is asepsis. Local anaesthesia of the entire needle tract should be insisted upon for the prevention of pleural shock. Excessive pressure within the pleural cavity should always be avoided and even positive pres-

sure is not entirely necessary for the production of effective compression. An important exception should be made in cases of severe hemorrhage in which bleeding persists till a marked positive pressure is established. Lung compression should never be employed in the presence of severe cardiac complications. The most frequent complication in the course of artificial pneumothorax is pleural effusion. It is variously ascribed to infection, to progressive irritability of the pleura due to trauma, to slowing of the lymph circulation in the compressed lung, to breaking of tubercles through the pleural surface, etc. It is estimated that this incident occurs in from fifteen to fifty per cent of cases. The exudate may remain serous, become mucupurulent or even pus, all of which adds to the gravity of the case and here a radical surgical operation is frequently indicated. The most serious complication is gas or air embolism, the onset of which is usually sudden and the minifestations may be from temporary unconsciousness to convulsions, hemiplegia, to gradual recovery or to fatal coma in from fifteen minutes to two or three days. It is supposed, according to Saugmann, that the fatal termination results from lodgment of air or gas in the coronary arteries or in the vessels of the brain. It is the opinion of many observers that this grave incident by a better technic and greater care can be avoided.

In Saugmann's experience between 1906 and 1910, 98 patients, 2,000 punctures—1 death; between 1910 and 1912, 63 patients, 1,200 punctures—1 death; between 1912 and 1914, 49 patients, 2,000 punctures—no death.

Forlanini—10,000 operations, gas embolism four times, two fatal. 1,058 cases treated by 22 American operators, the accident occurred three times in a group of 191 cases by three authors and in the remaining 876 cases by 19 operators, the accident did not occur in a single instance. Pleural reflex, pleural shock or eclampsia, as it is called by Forlanini, thrombi and other grave manifestations during or following the induction of artificial pneumothorax are all ascribed as of gas embolism.

Immediate and ultimate result of lung compression. Almost immediately following the induction of a pneumothorax the cough is increased, the expectoration becoming more copious as a result of expulsion of the secretion from the lung and in cases where cavities have been compressed, enormous quanties of muco-purulent or purulent material have been expectorated.

This, however, is soon followed by a diminution and nearly total disappearance of all expectoration. With collapse the temperature frequently rises due to a flooding of the lymphatics with toxins of the diseased lung, but this soon ceases, as the compression becomes more stable by a decline or disappearance of all fever, showing diminution or inhibition of absorption from the mixed as well as from the tuberculous infection. In a number of cases the heart's action also becomes greatly increased, especially on compression of the left lung, or distressing palpitation may follow if the heart is moved too quickly from its normal position. Here even dire results may follow. Compression is seldom followed by cardiac hypertrophy. The pulse almost always becomes rapid, feeble, hardly palpable, but with amelioration of the other symptoms the pulse is usually again normal. Nervousness may supervene incident to the procedure, the patient becoming pale, faint or showing an expression of fear. This, however, is usually only transient, the composure returning very rapidly. The injected gas or air in the pleural cavity may exert pressure upon the diaphragm, causing annoying gastric symptoms and in some patients a most distressing hiccough has been observed. In most individuals there is a temporary and slight loss in weight which is very soon followed by a reaction of gradual and perceptible increase.

The ultimate result of the induction of artificial pneumothorax after all is the bringing about of the formation of scar or connective tissue. This stands out distinctly in the healed or arrested cases and is the evidence of the efficacy of the treatment. Some patients almost from the very beginning of the compression express a feeling of general well-being noticeable by a marked improvement in health. The question is often asked how long the treatment should be maintained or how long the lung should remain collapsed—long enough to give nature an opportunity to form a firm fibrosis in the diseased pulmonary tissue independent as to time that may be required. A lung should not be permitted to resume functioning until assured that it is entirely healed; to achieve this the time of compression may be continued from one-half to two years and longer. Even after that length of time of collapse the lung when released will gradually resume its function. The pneumothorax must be maintained within certain limits of volume of compression so as not to interfere very materially with the free movements of excur-

sion of the healthy lobe which during the collapse of its mate is compelled to do compensatory or vicarious work. In far advanced or active and very chronic cases the compression if induced should always be maintained and the refillings at stated intervals should never be omitted.

It may be of interest to note the results of lung compression as observed by both American and European operators. The final or ultimate result is very similar in both countries. The result of 1,108 cases of pulmonary tuberculosis treated by 24 American operators corresponds very closely with the result of 224 cases treated by three of the leading European authors: American 21.7% of quiescent, arrested or cured cases as against 25.9% European. American 29.2% of improved and palliative cases, 31.2% European. American 49.1% of failures to induce lung compression, 42.9% European unimproved or dead.

Thoracotomy. Surgical operations other than pneumothorax for the treatment of pulmonary tuberculous disease (89).

(a) **Pneumonotomy. Incision and drainage of pulmonary cavities.** This procedure, in isolated cases, dates back for centuries, but the real attempt at drainage by surgical means extends to the time of the introduction of tuberculin as a therapeutic remedy. It was considered an established fact that the administration of tuberculin favored the production of a sequestrum about the walls of the tuberculous cavities which it was desired to eliminate. As many cavities empty themselves through the bronchial tubes without surgical interference the opening of cavities through the external thoracic wall has been little practice; besides, if an incision was made it usually left a pulmonary fistula which added greatly to the patient's discomfort.

(b) **Pneumonectomy. The removal of a part of a lung or of a complete lobe.** According to Murphy: "Pneumonectomy has been the triumph of surgical technic but clinical results have been extremely unsatisfactory and whilst the operation of removal of a complete lobe of lung has been successfully performed the number of deaths following pneumonectomy has been so large that the operation has practically been abandoned." Most operators now agree that in the surgical procedure for the obliteration of cavities by pneumonectomy the prognosis is extremely grave and a combination of all methods of rib resection which follow collapse and compression of the lung and ultimate scarring of the cavities with pneumonectomy is the only advised surgical method. We observe that pneumonectomy for the compression of cavities frequently is not a practical procedure; be-

sides grave danger accompanies the method; however, pneumonectomy may be advisable if the cavities lie very superficially. The only procedures of real value are those which aim to put the diseased lung at rest; namely, pneumothorax and thoracoplasty.

(c) **Pneumolysis.** An extra-pleural packing operation for cavity compression suitable for unilateral, chronic tuberculous cavities situated in the upper lobes, less applicable in lower lobe involvement. A subperiosteal resection of several ribs on the upper half of the thorax. The principle of the operation consists in an extra-pleural separation of the diseased portion of the lung by an artificially produced thorax window, removing from one or two ribs a piece about 6 cm in length and through this window with the index finger carefully separating the parietal pleura and filling the so produced space with a sterile non-absorbable material. The only severe difficulty encountered is the separation of the costal pleura, leaving the tissue wholly intact. Various substances have been used. Tuffier uses for extra-pleural packing or implantation sterile fat, Baer, paraffine prosthesis and Gwerder following the method of Baer observed that a hollow body is equally as efficient and uses a rubber ball or a rubber balloon with a tube attachment which allows of its inflation after it is put into place. This pneumatic plombing or packing is very simple, safe and non-irritating, adapts itself well to the irregular outline of the cavity and the pressure may at will be increased or decreased. If fat is used as a packing or plombage it may be used in the fresh state and in that event it may be taken from the patient or from a friend whose abdomen is well padded with fat. The fat so implanted is usually healed in about three weeks and beginning organization is already noticeable. Wilms is of the opinion that fat so implanted from one individual to another does not undergo changes. It may usually require from 300 to 450 grams of either fat or paraffine to completely fill a cavity.

(d) **Phrenicotomy.** Artifical paralysis of the diaphragm in the treatment of pulmonary tuberculosis, Stroutz 1911, Sauerbruch 1913. The lung is put to rest by placing the diaphragm in the position of extreme expiration. The treatment is proposed for severe one-sided chronic particularly cavernous lower lobe tuberculosis by severing the phrenic nerve in order to put the diseased lobe to rest. In cutting the nerve, the diaphragm ascends to an

expiratory position, the mobility of the lung diminishes, the lower lobe becomes compressed, lessening the danger of aspirating tuberculous material into the remaining healthy lung tissue; this has been confirmed both clinically and roentgenologically. The operation is simple and is usually done under local anaesthesia using a 1% novocaine solution.

(e) **Thoracoplasty—partial and complete.** Partial rib resection as practiced by Wilms and complete removal as advocated by Friederich, Marburg. A method for bringing about a lessening of the circumference of the chest and a lung contraction for the treatment of pulmonary disease, as recommended by Wilms, consists in the removal of pieces of rib from the posterior chest wall near the spine from 3 to 4 cm in length. A piece is taken out of every rib from the first to the eighth inclusive, some say to the 10th, and eventually at a subsequent time the same operation is performed on the anterior chest wall and by this so performed operation the circumference of the chest can be lessened from 6 to 8 or more cm or about three inches, permanently lessening the capacity, the elasticity and the excursion of the lung, bringing the diseased parts into close apposition, and favoring a healing tendency. The thoracoplasty of Friederich consists in an operation for extensive resection of the ribs on the affected side from the second to the tenth inclusive in extensive one-sided tuberculous disease. The ribs may be removed near the mediastinal fixation or from the posterior axillary line to near the sternum. The patients selected should be between the ages of 15 to 40, as beyond the latter age the resistance to overcome the shock of an ordeal of such proportions necessary is usually wanting. Extremely anemic individuals as well as those who are suffering from recent tuberculous involvement of other organs must be excluded. In cases of very active tuberculous disease running a rapid course in which there are dense pleural adhesions and which would not justify an attempted pneumothorax, extrapleural thoracoplasty, either partial or complete becomes an operation of choice and cases favorably influenced by these operations show a collapse and obliteration of the cavities, subsidence of the fever, lessening of the sputum and general improved condition. These operations on the thorax should be done by removing the ribs either whole or in part subperiosteally and without injuring the parietal pleura. In some cases it may become necessary to combine thoracoplasty and phrenicotomy,

especially in upper lobe tuberculous disease, so as to avoid the aspirating of tuberculous material into otherwise healthy tissue.

Most surgical operations on the thorax, artifical pneumothorax, pneumonectomy, phrenicotomy, thoracoplasty all aim to immobilize the lung, to put it at rest, to keep it quiet and compressed for a sufficient length of time to bring about an arrest and healing of the tuberculous process. Paradoxical as it may seem, another thoracic operation is advocated in pulmonary tuberculosis which aims at a decompression to give the lung more motility, more power to expand, if the disease is already established. This also favors an arrest or healing of the tuberculous process; if not already established, a freer lymph and blood flow inhibiting a beginning disease. This operation known as chondrotomy is applicable only if the involvement is in the apex of either upper lobe.

(f) **Chondrotomy.** Freund's operation, in apical tuberculous disease, consists in a resection of the cartilage of the first rib and widening of the aperature. He is of the opinion that in some cases of beginning tuberculosis the abnormally short first rib and early ossification of the cartilage bring about a shortening and a consequent stenosis of the upper aperature; this if it is not the direct cause of tuberculous disease, owing to loss of respiratory movements, furnishes in all probability a predisposing factor, and a separation of the cartilage and an enlargement of the upper aperature gives a better aeration and a more free lymph and blood circulation through the apices. This theory has many followers but also many opponents, the latter maintaining that ossification of the cartilage is not primary but a secondary condition, is really an attempt of nature in active tuberculous disease at lung immobilization and rest and that for this reason demobilization is contraindicated. However, there is no doubt that both Freund and his pupils, Hart, Harras and many other operators have proven the excellent result following the operation in selected cases.

CHAPTER 21

ASSOCIATED THERAPEUTIC MEASURES IN PULMONARY TUBERCULOSIS

(A) CHEMOTHERAPY IN TUBERCULOSIS

A therapeutic agent which, if incorporated into the body of the tuberculous individual, would have no deleterious effect or influence upon the normal body cells or tissues would be perfectly harmless but would have a definite and decided inhibitive and destructive influence on the tubercle, tuberculous tissue and the tubercle bacillus has been the wish, and its discovery the fond hope of the clinicians and the aim and the object of research of the laboratory diagnosticians. A remedy which, under most favorable conditions, could be brought into close contact in the human body with the living germ or virus, in short, a therapea magna sterilisans in the Ehrlich sense. Up to the present time we are not familiar with any such acknowledged or approved remedy, but that such a preparation may be within the near possibilities of experimental medicine can, judging from the indefatigable work being done, be accepted.

The use of chemical substances for the inhibition of bacillary growth within the human or animal body is not of recent introduction. Soon after the discovery of the tubercle bacillus as the etiological factor of the disease this was attempted, and we learn that as early as 1890 Robert Koch had shown that gold cyanide will inhibit bacillary growth in the experimental animal in a dilution of 1 to 2,000,000. Luton, a French dermatologist, in 1894 first suggested the use of copper salt preparations as a therapeutic agent in the treatment of cutaneous tuberculosis. At the International Tuberculosis Congress in Rome in 1912 the use of copper salt solution as recommended by Prof. Finkler was much discussed. He and his co-workers tried experimentally the various salts of copper, the sulphate, chloride, and tartrate in definite strength solutions, but the best results were obtained from a complex copper preparation known as Leucetyle, a combination of copper, lecethin and cinnamic acid, and lecithin in

combination with iodo-metheline blue. Most of this work was done after Finkler's death (89) by the Countess v. Linden at her laboratory. It was then suggested that the supposed favorable influence of these complex copper salts on the tuberculously infected experimental animal be tested on the human, and Meissen, a clinician, applied it in the treatment of pulmonary tuberculosis, and Strauss, dermatologist, in a number of tuberculous skin lesions. At the laboratory of Prof. H. Gideon Wells of the University of Chicago, Dr. Lydia M. DeWitt and assistants have done a great amount of experimental work along these lines, using various chemicals and chemical combinations. The result of these carefully conducted experiments were not as hopeful as we were lead to believe from the above mentioned earlier work.

The chemicals generally used by most laboratory workers were besides the copper salts mentioned above, the acetates, oleates and to some extent colloidal copper. The introduction of drugs into the animal organism was done in all conceivable ways—by feeding, by intravenous, subcutaneous, intramuscular and intraperitoneal injections and injections into the anterior chamber of the eye. The results of all this experimental work on the previously infected animal were about the same, namely, that in no case was any positive inhibitive effect on the tuberculous process noticeable. The observations with these various copper salts admit the following conclusions: if the entire body, the blood and fluids circulating throughout, could be made to carry at all times one part of copper to 100,000 parts of body weight, we might be able to inhibit the growth of the tubercle bacillus and perhaps eventually also to kill it; copper is of no particular value in the fight against tuberculosis, in fact, its use is unjustifiable, is even dangerous.

Of added interest in Chemotherapy is the fact that many water soluble dyes possess the property of penetrating the tubercle bacillus in tuberculous-tissue. This had already been studied by Ehrlich and his co-workers at an early date, and vital stains or such dyes as can be introduced into the living animal without material injury to the tissues or cells are now extensively used for experimental purposes. Paul A. Lewis and Robert B. Krause reported their observation with trypan red to the effect that if this dye is injected into a living tuberculous animal it becomes concentrated in the necrotic portion of the tubercle. Other in-

vestigators report similar properties of a number of dyes including trypan blue, isamine blue, pyrrhol blue, iodo-metheline blue; many others have been used, including combinations of trypan red with iodoform and with Thymol, Eucalyptol, Guaiacol, Iodin, etc.

According to DeWitt metheline blue is the most classic example of a vital stain or dye now in use in chemotherapy. The permeability of tissue cells to drugs and dyes has in recent years been extensively investigated and it was proven conclusively that in the tuberculous lesion there exists a special affinity or specific attraction for some of these drugs, an affinity inherent in the caseous portion of the tubercle; both trypan red and iodine, when injected into the tuberculous animal, are found localized in the necrotic portion of the tubercle, but so far as observed they have no favorable therapeutic action on the tuberculous process. A combination of the salts of gold with cantharadin as Aurocantan has given much promise in chemotherapy. Cantharadin when administered causes a local reaction about the tubercle and other inflammatory foci, and this fact may be utilized in using cantharadin as a vehicle in carrying the gold into the tuberculous tissue. Both auric and aurous salts seem to exhibit a specific attraction for tuberculous tissue. The property of permeability of the tuberculous lesion by these chemicals is due to the fact that these tissues all behave in every respect like simple non-living colloids permitting crystallin bodies to diffuse readily but being little or not permeable to non-crystallizable or colloid substances, simply a process of osmosis or diffusion. Loeb first showed that a tuberculous lymph gland takes up relatively more iodine from the blood than any of the other organs but that the caseous material of the gland takes up many times as much as the remaining still normal gland tissue.

Many chemical substances and various salts and combinations have been used and tried in experimental chemotherapy, the mercuric and mercurous chloride, iodide, salicylate, succinamate, etc., arsenous and arsenic salts and many others, and all have been found worthless, unreliable and dangerous.

Lydia M. DeWitt on "Copper in the treatment of tuberculosis" makes a terse statement which seems to apply to nearly all the other chemical substances used. "Those therapeutic measures given just before or at the time of remission of the active tuberculous disease may be found a favorable time to be

regarded as an apparent improvement and may be even so heralded as a certain curative agent, whereas, if given just before or onset of a period of exacerbation these same measures may be considered as worthless or injurious."

Organic Acids in Chemotherapy

Many diluted organic acids, lactic, phosphoric, malic, etc., dissolve tubercle bacilli as is true in the preparation of the partial antigens according to Much, Deyche and Leschke.(190) In culture media it has been proven that diluted lactic acid kills and dissolves tubercle bacilli and bacilli circulating in the blood or in recent tuberculous inflammatory areas can be affected by intravenous injection of diluted lactic acid. It may be indicated in such cases as are usually early recognized by experts and which in general practice are usually referred to as neurasthenia, anemia, cardiac palpitation, gastric disturbances, dysmenorrhoea, rheumatism or to other prodromal signs. It may also be used in bronchial gland tuberculosis and in cases of mild and closed pulmonary disease, that is, while the disorder is still circumscribed. Phthisical cases are not suitable because the diseased area cannot be influenced by the blood current, and then again the lytic action may be so pronounced that the body can not cope with the effected bacteriolysis. It may be possible that the acid has bacteriolytic action on the bacilli circulation in the blood current and also on the bacilli in the pulmonary tissue, which the blood stream may surround, and then again it may also be possible that the action of the diluted lactic acid causes a dissolution of some of the red blood corpuscles liberating the autoimmune bodies, on the theory advocated by Carl Spengler. In selecting cases, individualize, using a 1% lactic acid solution injected once or twice weekly, finally once a month. In beginning cases it may give most promising results.

Iodine in Chemotherapy

When iodine in the form of the tincture or its salts is administered to the human body in whatsoever way it readily makes its presence known in the blood. Next to the blood the normal kidney is most permeable, containing as much iodine as the blood itself, and during active secretion even more. Iodine is very rapidly and quickly eliminated through the kidneys and its presence in the urine is demonstrable in a comparatively short time after its incorporation. The blood always contains more iodine than any tissue or organ of the body,

normal or abnormal.(85) The liver and spleen usually only contain about 1/3 as much, the lungs a little less; the lymph glands, however, take up more than either the liver, spleen or lungs; muscle tissue is usually not very permeable, usually containing only about 1/10 as much as the blood; however, necrotic tissue, even necrotic muscle tissue, is very permeable, containing as much iodine as the blood itself, and the same applies also to an inflammatory exudate. As stated above, this is due to the fact that colloid masses permit crystalloids to pass readily, but nearly completely inhibit the passage of colloids. Based upon these well known facts I began a number of years ago, in many forms of tuberculosis, the administration of tincture of iodine in enormously large doses, with most gratifying and encouraging results. The human body is most tolerant to unlimited doses of the tincture and it can be given for months even years without the least disturbance. Iodine tolerance can not be satisfactorily tested on the experimental animal, but its long use now for many years has convinced me that the human body is tolerant to excessively large doses.

Practically some years ago, by a mere accident, I learned more of this tolerance. It came about in this way. I was consulted by a colleague concerning a woman who was operated on for gall stones and who about a week after the operation developed a severe cough which became so persistant and the expectoration so copious as to attract the attention of the attending physician, who upon examination of the sputum found it tubercle bacilli positive. My first thought on hearing about the case was that it was a miliary disease and I directed that she be given 20 drops of the tincture of iodine in milk three time a day. As months passed I was informed from time to time that she was steadily improving and was gaining in weight and as I now became more interested in the case, I desired a consultation with a view of making a physical examination. This proved that she had a right sided old tuberculous pleurisy. I now also learned that she had misunderstood the general directions and in place of taking the tincture of iodine 20 drops three times a day, had taken twenty drops every two hours, and appearing in such general good health, I directed that from then on she take twenty drops every hour, which she did, continuing this dosage for many months. Repeated sputum examinations were negative and most particularly a distinct gain in weight was noticeable.

Many cases of tuberculous adenitis, bone and joint disease, psoas abscesses and chronic pulmonary tuberculosis have been on an iodine treatment persistently for months and years with most gratifying results. Iodine in the form of the tincture may safely be administered in extremely large doses and for very long

periods of time without anticipating any deleterious effects. Iodine is perfectly innocuous and non-irritating to the gastric mucosa if given in its proper vehicle, good wholesome milk. Further observations have convinced me that beneficial and lasting results follow the ingestion of iodine only if large doses are given; small doses are not dependable. When iodine is administered, its presence can readily be demonstrated in the various fluids of the body, both excretory and secretory, but within a comparatively short time nearly all, excepting perhaps a slight trace will have again disappeared from the body. Therefore, it should be given persistently, often repeated and in fairly large doses to keep the tissue saturated and to inhibit bacterial growth. For some time I have been administering the tincture in 10 to 30 and more drops in milk at intervals of 4 or 5 hours or at breakfast, luncheon, dinner and at bed time. As iodine when administered with milk is perfectly harmless and innocuous to the tissue and cells of the body, but is largely destructive and inhibitive to all microörganisms, its use can not be followed by any deleterious results to the human economy.

It has repeatedly been observed that if to a tuberculous individual intensive doses of tincture of iodine have been administered for some time, his tuberculin sensitiveness disappears, so far as it concerns a rise in temperature. This fact seems to depend upon the reduction or lessening of the temperature, producing tuberculotoxic substance in the body by virtue of the iodine ingestion.[1]

(B) HELIOTHERAPY—SUNBATHS

Light Treatment. (Sun Light, Arc Light, Quartz Lamp, X-Ray, Phototherapy, Radiotherapy, Roentgenotherapy, Etc.)

Historical Note: Heliotherapy was well known to the ancient Greeks and its method of application in those times is well described in the works of Herodotus B. C. 430.(18) He mentions: "To bathe in the sun is necessary to increase and strengthen the muscles of people in need of such treatment. The direct rays of the sun are favorable in winter, spring and fall but not in summer to weak and feeble persons." Hippocrates, it is said, exposed his patients to the direct rays of the sun, and Celsus recommended sun baths as a potent remedy in epilepsy and in

[1]In administering Tincture of Iodine the patient should always be under the care and supervision of the attending physician (87). Very exceptional patients are intolerant to the ingestion of Iodine. In such instances, the use of the drug must be discontinued, most patients, however, are tolerant to very large doses. Patients who exhibit much nervous disturbances, increased rapidity of the heart's action or hyperthyroidism during the Iodine treatment are not suitable and the drug should be discontinued.

disease of the abdominal organs. We observe that, in prehistoric times, prehistoric man directed his caves all towards the east or south, most presumably on hygienic principles. The sun was worshiped by the Egyptians in Rhea, the Sun God, by the Persians and Greeks in Heliō and in Sol by the Latins. The use of sun baths was extensively practiced by the Greeks in their Heliosis or resting places about their homes, below the domes of the temples or in the sands. Excavations made in the old temple of Aesculapius in Epidaurus exhibit a long gallery with a south exposure connected with rooms or wards for the sick. In ancient Rome there was a Solarium in every home, and pictures from Pompeii show many examples of the inhabitants, who were wont to take their sun baths on the roofs of their homes or in terraced places. All Thermos were provided for sun baths.

We find nothing more recorded about the sun as a health given agent from about the second century until the second half of the eighteenth, when Faure in 1774 reported to the Royal Academy of Medicine his observation on the "Use of heat from sunlight in the treatment of ulcers"; LePeyre and LeCounte, 1776, increased the action of the sun by passing its rays through lenses, reporting the cure of ulcers and even of carcinoma; Bertrand, 1799, presented a dessertation and essay before the Faculty of Medicine, Paris, on "The influence of the sun's rays on diseased parts of the body." Heliotherapy received its first definite recognition 100 years ago when Lobal in 1815 in the Journal f. pract. heilkunde stated that he recommends insolation in all cases of indisposition in which the vegetative system of the organism has suffered, when the extremities are cold, for general weakness and in an inactivity of the lymphatic system; such treatment is indicated in gout, rheumatism, dropsy, diarrhoea, intestinal disturbances, imperfect functioning of the skin, in nervous disturbances, but is contraindicated in acute inflammations, active lung affections, hemoptysis, congestions, etc. The French school at Lyons was the first to apply heliotherapy in the treatment of tuberculous arthritis, and Bonnet, 1845, in his treatise on the diseases of the articulations recommends insolation of the knee joint as a matter of choice. Lebert, 1849, "Practical treatise on scrofulous and tuberculous diseases," advocates the use of heliotherapy in chronic joint disease. Turek, 1852, called attention to the efficacy of sun and light baths and gave specific directions for their use.

Bonnet's idea was further advanced by Ollier and subsequently by Poncet applying heliotherapy in the treatment of tuberculous joint diseases and a pupil of the latter was induced to write a treatise on "Insolation in the treatment of articular tuberculosis," reporting four cases which were very much improved by this method of treatment. Previous to 1911 Rollier knew nothing about the work done in heliotherapy by the pupils of the French school at Lyons and the work in heliotherapy was done and the results achieved by him independent and without any knowledge that such was ever attempted before. Rollier applied heliotherapy in the treatment of tuberculous disorders most extensively in every stage and in every form, but especially in bone, gland and joint disease for many years with most gratifying results. To Rollier (assistant to Prof. Kocher), who (18) in 1903 instituted the first establishment for the cure of surgical tuberculosis by means of the sun's rays at high altitude, (at Leysin in the Vandoises Alps, Switzerland, 4200 feet above sea level), much credit is due for bringing heliotherapy prominently before the medical profession as a most efficient measure in the treatment of tuberculosis, not only in surgical where it finds its greatest application but in all other various forms, if only diligently used. The demonstration in 1899 by Finsen of the effect of red rays upon variola and ultra violet rays upon lupus was an epoch making event in the application of the sun's rays as therapeutic agent, and since that time much has been done in heliotherapy in giving a scientific explanation of the effect of the rays upon the organism. Much work along this line was done by Roux, Arloing, Winteritz, Panwitz, Schroetter and others and the work was all concentrated upon the penetrability of the solar or light rays into the human body and upon the possible effect this would have upon the pathogenic microörganisms.

Heliotherapy in Tuberculosis

In order to know what healing effect the application of the sun's rays will have on the tuberculous process we must briefly consider, first, what particular rays possess a special inhibitive action on the tuberculous process, second, by what special method or technic can this object be accomplished, and third, what physiological manifestation does this curative process bring about.

As to the varying intensity of these rays, we note that the

difference in intensity of the ultra violet rays between a high and a low altitude is that it reaches its maximal in winter and becomes less as the warm season or summer approaches.

Physical meaurements have proven that the ultra violet rays are the ones which in heliotherapy are the beneficial rays, and the question arises in which way and under what biologic conditions they become active and evidence their presence. It is known that pigmentation is brought about by these rays; we also know that pigmentation predisposes a healing tendency, and is an index to a prognosis; and it has also been proven that the appearance of pigmentation evidences the presence of ultra violet rays in white light.

The Effect of Heliotherapy on the Human Body

Following the use of the sun's rays on the body, there is an increase in the number of erythrocytes, a decreased leucocytosis, an increase of the hemoglobin content, and with pigmentation the lymphocytes increase proportionately. This improvement is evidenced from the very beginning of the treatment. In closed tuberculosis the leucocyte count is below 10,000, but in open adenitis an increase is present, an increase of the polymorphonuclears and a lessening of the lymphocytes. With healing a change in the curve is observed, the polynuclears decrease and the lymphocytic curve increases. This increase of the lymphocytes may be the result of active hyperemia due to the stimulating effect of the treatment. Insolation produces vasodilation of the blood vessels and through extensive hyperemia of the integument a decongestion of the internal organs and increased textural changes in the derma are brought about, and an increase of body energy from the absorption of light in the presence of hemoglobin and increase of the ozydizing changes, favor an exchange of gases between the blood serum and the tissue cells. The peripheral blood by means of the activity of the short waved rays is altered, is energetized, giving up this energy to the inner tissues and the increased energy changes the oxy to methemoglobin. Light favors the free supply of oxygen to the needy tissue cells. It has also been shown that the ultra violet rays have a decided effect on respiration, by lessening the frequency of breathing for the time being, but increasing the fullness of the inspiratory phase.

The Effect of Insolation on Individual Organs and Tissues of the Body.

Heart. The activity of heliotherapy upon the heart is manifested by a decongestion of the internal organs and a lessening of the blood pressure, most likely by the direct influence of the ultra-violet rays upon the nervous system.

Blood Pressure. The lessening of the blood pressure is a result of dilatation of the peripheral vessels by means of the direct influence of the rays upon the vaso-motor dilators. The rays are pain relieving and sedative in their action.

Kidneys. The action of the rays upon kidney functioning is manifest by increased diuresis.

Mind. The action of the ultra-violet rays upon the mind of the tuberculous is particularly noticeable and the mental effect is the hopeful spirit, the buoyancy, the increased activity and increased working capacity and above all an increased feeling of well being.

Metabolism. Another effect of the ultra-violet rays is upon the ferment action accompanying metabolism, favoring dissociation which increases the oxygen consumption with a consequential increase of nitrogenous waste.

The Posological Consideration in Heliotherapy

Here, as in all phthisotherapeutic measures, exact dosage is necessary. The indications for dosage vary very much, different clinicians recommending different amounts. Malgal does not exceed the time of heliotherapy to 20 minutes, whereas Rollier, on the other hand, extends its application to as much as eight hours. The beneficial influence of the sun's rays on the body must be judged by the subjective sensations, pulse, temperature, blood pressure and pigmentation of the skin, the intensity of the latter being proportional to the favorableness of prognosis. The three important factors to observe and to consider are (1) the light of the sun as a whole, (2) the reaction of the various organs to heliotherapy and (3) the special reaction of the different disorders to the treatment.

(A) **As to light.** The light of the sun is composed of heat, light and chemical rays, is subject to changes, as to locality, intensity, etc., depending upon whether the treatment is to be considered at the seashore, in the valleys or in a high altitude, hence a dosage in sun's rays must be elaborated which is suitable to these various conditions. For instance, the treat-

ment at the seashore must consider the variable climatic conditions. The direct insolation or exposure to the sun's rays is lessened in intensity from the high to the low level and the direct opposite with diffuse day light. The direct rays traveling from above down do not lose in force proportionally as much as the indirect rays gain, and considering the reflection from the washed surfaces, the surrounding shores, the cloudy days and moist atmosphere, it becomes very evident that a constant dosage must be desired and the technic understood. A cold sunbath below body temperature may act as a hypertonicum and may lead to very unfavorable results, while a warm sunbath above body temperature may result most favorably.

(B) **The reaction of the sun's rays on various organs.** We find that the sun's rays act locally in two ways, physiologically and therapeutically, namely, bacteriocidally, increasing phagocytosis, favoring fibrosis, producing scar tissue and relieving pain. A cutaneous erythema or what is known as dermatitis solare is to be especially guarded against, but pigmentation is desired. Favorable reactions, like a lessening of the amount of sputum or the return of a normal bowel movement after heliotherapy are the objects desired, and unfavorable reactions, like vomiting following the application of the sun's ray to the abdomen for the treatment of tuberculous peritonitis or enteritis show that the treatment is not suitable and may do much harm. A favorable reaction is evidenced by a feeling of well being with an increase of body weight; without these, the dosage is faulty. The observation of the subjective sensibility is only of relative value, but the registration of the pulse, temperature and respiration is of unusual importance. Generally after the bath there is a slow rise in all three, which after a while is again equalized. Continuing in the bath, both pulse and temperature remain usually below the initial bath, but the respirations remain as high as in the beginning, gradually dropping to the level indicated before the bath. In the beginning of the treatment deviations from this rule may be present but they do not last. If they do not disappear the dosage is either not well selected or the individual possesses an intolerance to the sun treatment. Pulse frequency, respiration and temperature alone can not be considered; the question of the reaction immediately after the sun bath must be taken into account.

The blood changes indicating a favorable influence in helio-

therapy are an increase in the number of erythrocytes and an accompanying increase of hemoglobin, a slight lessening of the white corpuscles with the presence of eosinophiles. An increased leucocytosis usually indicates either a retention of the tuberculosis toxins or a more rapidly progressing morbid process and if present is generally a sign of caution. The tuberculin test may be applied to give an indication as to the result of the treatment, the reaction being negative if the therapy is not well tolerated by the organism, becoming positive if conditions are more favorable. It must be borne in mind that heliotherapy when applied to pulmonary or peritoneal tuberculosis requires greater care and caution than when used in the treatment of lupus or certain tuberculids.

(C) **The special reaction of the different disorders to the treatment.** The most favorable conditions for heliotherapy are bone, joint and skin tuberculosis, but with a careful technic good results are also achieved in intestinal and urogenital tuberculosis in mediastinal gland and pulmonary disorder but they require greater caution and closer observation. In children, if the disease of the tracheobronchial glands has extended beyond this region and has actively invaded pulmonary tissue, the use of the treatment by heliotherapy is contraindicated. It is also contraindicated in hemorrhagic cases.

Technic and Clinical Results

(A) Exposure to the solar rays. In using the rays for healing or curative purposes it is necessary that we begin with a very light or but little exposure, gradually exposing the whole body as far as the head to the action of the rays. Beginning at the extremities, little by little, gradually extend the exposure to the abdomen and ultimately to the chest with a one to three minute exposure, and if no unfavorable indications arise, increase daily by three minutes; this is in internal disease. In external or local disorder the rise is more rapid to 5 minutes daily. The best results are attained by exposure to a temperature between 86 and 104, and exposures above and below these points may be well tolerated by vigorous patients but must be applied a very limited time. The treatment must extend over long periods of time in some cases, as in gland tuberculosis, perhaps two or three months, while two or three years may be required in Pott's disease. The treatment must be continued until the symptoms have

completely disappeared, otherwise a quick return of the disorder results.

According to Kisch and Graetz, (212) bone and joint tuberculosis with fistulae are most favorably influenced by heliotherapy, requiring on an average about one year's treatment. Fistulae of the soft parts require about from 4 to 6 months. The cure of tuberculous fistulae under heliotherapy is not due to an encapsulation of the mycotic or fungoid substance nor to a burying of the tuberculous disease but to a contraction of the tuberculous mass and the formation of new scar tissue. Hermann v. Schroetter, Vienna M. M. W. 1914 No. 21 says: "Insolation in combination with fresh air treatment is the surest therapy in surgical tuberculosis and can never do any harm. Heliotherapy should find its greatest applicability and extensive use in childhood. By means of its early use to free the system from diseased influences, to bring about a healing of local disorder, insolation becomes a prophylactic measure for the future healthy growth and development of the child. The result in the main is dependent upon the unlimited duration of the treatment. The tendency towards tuberculosis and the limited capability to pigment formation stand in relation, possibly in combination, with changes in the internal secretions. The respiratory phase and character of breathing are stimulated by insolation. All are agreed that insolation must be a very gradual process beginning by applying the rays to the lower extremities and gradually extending the treatment upwards, covering all of the body as far as the head. In all cases of arthritis, Pott's disease, fixation and immobilization is essential and like all other means it is not a cure, it is only a most valuable aid in therapy."

(213) C. Arndt admits that Rollier's excellent curative measures can not always be attained, and that in the valleys the result of heliotherapy is not as favorable as it is at high altitude. Even in the treatment of tuberculosis at high altitudes the healing is slow, very slow, but nevertheless an attempt at heliotherapy should be made in the valleys and solariums and sun bath facilities should be established in connection with every tuberculosis sanatorium, and if the institution is not located in the mountains, results may be achieved by having recourse to the roentgen ray or the quartz lamp in lieu of heliotherapy in surgical tuberculosis. In all cases that are favorable for the treatment and if it

can be extended over long periods much good may be accomplished.

R. Ridard, on the other hand (211), takes issue with the opinion of the enthusiast who maintains that all cases of localized bone, joint or gland tuberculosis are curable by means of heliotherapy and without surgical intervention. Without underestimating the value of fresh air, baths and insolation in surgical tuberculosis, orthopedic treatment is most desirable, and no rule can therefore be laid down. The treatment by insolation must be adapted to the form of the disorder, the place or location, the time, the complications, the age and the social conditions of the patient, and it is now admitted that it is only a valuable adjunct in the treatment of surgical tuberculosis, exceeding by far medicinal therapy. The assumption that surgical tuberculosis is only a local affair and requires only local measures in its treatment is no longer tenable; on the contrary it is preeminently a generalized disorder requiring in addition a dietetic, hygienic, and medicinal treatment, with rest and heliotherapy of the entire organism.

In our country heliotherapy as a measure for inhibiting, arresting or curing a tuberculous process has up to the present time not received the full attention it deserves. At the sanatoria situated south west amongst the mountains the instituting of the solarium should be a regular program and this should be attempted even at the sanatoria situated in the low lands. At low altitude the sun treatment may not be quite equally as effective in a given time, but with prolongation of the time most efficient results may be achieved. That not sufficient interest is taken in heliotherapy in our country is quite evident when we note that amongst all the papers offered at the National Tuberculosis Association at their annual meetings, beginning with the first meeting in 1905 and up to and including 1919, not a single one was ever presented on heliotherapy. It is equally of interest to note that at the meeting of the International Congress on Tuberculosis held in Washington, D. C., in 1908, with the exception of a paper on heliotherapy read by Rollier of Leysin, not a single other paper on this important topic was offered. Of the value of this method of tuberculosis treatment there can be no doubt, and it behooves us as clinicians to take advantage of this form of medication and to recommend in our large country the establishing of solaria where many cases which are suitable for this

method of treatment can have their tuberculous processes arrested.

In this country, the first systematic application of the sun's rays with the open air treatment in surgical tuberculosis according to the method of Rollier was attempted in 1914 at the J. N. Adam Memorial Hospital (168), Perrysburg, N. Y., under the supervision of Dr. John H. Proyer. This treatment was found so successful there that since that time its use has been attempted at various sanatoria throughout the country.

This Solarium in New York state is situated at a height of 1650 feet above sea level, and the results of insolation at this

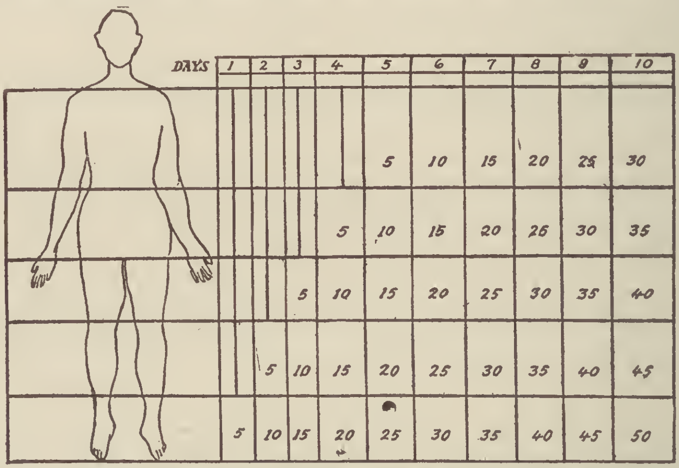

Fig. 40. Schematic diagram of Insolation. Showing the progressive exposure of a patient to heliotherapy. (After A. Rollier.) (From reprint by Drs. Clarence L. Hyde and Horace La Grasso, Perrysburg, N. Y., 1917.)

elevation are found to be nearly as efficient as that in higher altitudes. With but slight modification the technic used there is very similar to that applied at Leysin by Rollier. Usually the radiation is by direct exposure to the sun's rays, that is during bright days; to continue the treatment, however, during the days when there is no sunshine, artificial ultra-violet rays are used by means of a mercury vapor lamp. As a general rule, no attempt at radiation is made until after the patient has been admitted at least three days, and no insolation is attempted during the hottest days in summer as it is found to be too depressing, too fatiguing during this period. The dosage of insolation must be a graduated and gradual one; it usually is given according to the following rules, beginning with a most limited bath the first day and adding each day a little more exposure.

First day: The patient is dressed in a fabric or flannel garment according to the season, and the head and eyes are protected, the former by a linen cap, the latter by means of colored glasses. The feet are exposed and bathed in the sun's rays for five minutes three or four times at hour intervals.

Second day: The feet are insolated ten minutes and the legs from ankles to knees five minutes three or four times at hour intervals.

Third day: The feet are insolated for fifteen minutes, the legs from ankles to knees ten minutes, and the thighs five minutes three or four times at hour intervals.

Fourth day: The insolation of the previously exposed parts is increased by five minutes and the abdomen is exposed five minutes three or four times at hour intervals.

Fifth day: The insolation of the previously exposed parts is increased by five minutes and the chest is exposed five minutes three or four times at hour intervals.

Sixth day: The patient is now turned on his abdomen if conditions are favorable, and the same course is prescribed as in the previous five days.

It may be advisable to insolate the body both front and back the same day. Here then we may begin by bathing on the first day the anterior surface of the feet for five minutes and perhaps an hour afterward the posterior surface for five minutes, each twice a day at hour intervals. The second day expose both the anterior and posterior surfaces of the legs from the ankles to the knees for five minutes and the feet for ten minutes, and proceed similarly to exposing the body as given above.

The insolation may be continued, as is outlined in the appended diagram, until the baths are given daily for three to six hours. The chart here given shows the insolation for ten days. If it is desired to continue the same plan for more days, say up to fifteen, then we continue as in the preceding plan, adding five minutes more to each part exposed, so that on the fifteenth day the feet would be exposed for seventy-five minutes, the ankles and knees to seventy, etc., and after this time begin the day with a complete body bath.

Although heliotherapy is most serviceable in surgical tuberculosis, tuberculosis of bone and joints, the beneficial influence of the sun bath, the ultra-violet rays, on all the other forms of tuber-

culosis including pulmonary, if the process is not too active or rapid, must be admitted.

(C) HYDROTHERAPY IN PULMONARY TUBERCULOSIS

A healthy and vigorous individual with good physiologic power of heat production will find the cold morning bath a most wholesome and delightful measure (162). Such an individual on rising in the morning will get into a tub of cold water, take what is known as a dip, or remain in it from one to five minutes, dry himself and with a coarse towel thoroughly rub the skin till it begins to glow; this is called the reaction. Upon being dried and dressed he experiences a fine feeling of skin warmth, a most pleasant sensation; the cheeks are pink, he looks and feels well, eats a hearty breakfast and begins his day's work with vim. A sick and weakly person seeing this reaction in the healthy individual naturally becomes much impressed with the supposed value of the cold bath or spray. As a rule he does not seem to comprehend the difference in their physical make-up; in such an individual, try as he may, the cold bath is not followed by a vigorous skin reaction, but on the contrary, it has a most decided depressing effect, and whereas the vigorous person will look forward to his daily morning cold bath with a feeling of delight and enjoyment, the enfeebled and sickly soon begin to show distinct signs of dislike for it, complains that the bath is followed by headache, shivers, after the bath, and the nose and lips look blue, complains of rheumatoid pains, shows a nervous fear with the approach of morning at the thought of the cold bath. An active pulmonary tuberculous individual is generally a weakly one, possesses no power of reaction and in place of being invigorated by the bath becomes much depressed; hence the cold bath, plunge or shower should not be advised; on physiologic grounds alone the cold bath for the tuberculous is contraindicated. Being tucked up in a warm bed with sufficient blankets to maintain a uniform body heat, perhaps sleeping out-of-doors in the open, inhaling the cold air, all of this he greatly enjoys. Then, being in a warm bed, he is suddenly asked to leave his warm quarters to plunge or be plunged into a cold water bath. If there be much fever the cold bath may bring about a drop in the heat curve, but the depressing effect of the bath, the feeling of discomfort is very great. A daily sectional cool bath or sponge with the patient

remaining in bed, sponging first one limb, drying it and putting it under cover, then the other, next the abdomen, then the chest, the back, the arms and last the face, as usually done by a nurse or attendant, is a most desirable treatment, however, for the active tuberculous the daily warm or hot bath is the most effective hydrotherapy. Even for the tuberculous who is still strong the full or half bath in warm water is preferable to the cold, and the favorable influence of the cold bath on the fever curve can not be compared to that of the warm bath. We see that in the application of hydrotherapy individualization becomes a most important element. The variation of a few degrees of temperature in a bath may be sufficient to act as a stimulating tonic to the skin of a given individual whilst the same degree may become quite irritating to another. It is best always to give the bath in the morning at the time when the fever is absent or is at its lowest point. In febrile cases a warm bath given every third day will often tend to greatly reduce the temperature and in those suffering from night sweats a morning or evening bath or cold plunge followed by an alcohol rub will often inhibit these subjective symptoms. In the matter of giving baths, either cold or warm, the tuberculous patient's comfort must always be considered and not the individual taste of the attendant or nurse or perhaps of the physician in charge in whom the cold bath may act as a vigorous stimulant; here as in the treatment of tuberculosis in general, good judgment must be used and common sense applied. The patient immediately after the bath should be put to bed and remain for some time so as to avoid what is known as shock. With increase of body energy the cold cloth application may be applied to the naked body from the shoulders down, the patient standing, the attendant rubbing the body vigorously through the wet sheet; this is then followed by a dry cloth application and very gentle rubbing, after which the patient is put back to bed to remain lying for about one hour. A warm foot bath on retiring often gives very prompt relief from headache, restlessness, insomnia, and sweaty and coldish feet. A short, slightly cold sitz bath with abdominal massage during or before the bath often gives prompt relief for stubborn and chronic constipation.

Hydriatic measures much in use at many tuberculosis sanatoria, particularly in Europe, are cold water dressings according to the directions of Winternitz. Clean, absorbent roller bandages

of suitable width are wrung out of cold water and applied to the chest in the figure of 8 form. Beginning posteriorly at the right axilla the bandage is passed over the left shoulder back under the arm, to the opposite shoulder under the arm back to the starting point, a second layer is applied in a similar manner, then continue the bandage over the anterior and posterior chest down to the 8th rib in the axillary line. A dry roller bandage is now evenly covered over this, and all may then be covered by a tight fitting vest.

Nervous individuals and those with a very sensitive skin usually complain much if cold water dressings are applied according to this method; here vigorous rubbing of the skin previous to the cold dressing will often obviate the discomfort. This form of bandaging is best applied just before retiring. It may, however, also be applied during the day, in febrile cases once in three hours and in the afebrile once in six hours. It is advisable always to give the skin a thorough rub down before applying the dressing. The chief value of this cold bandage depends upon its beneficial influence on the respiratory organs. The cold application at once produces a general feeling of warmth and by means of the tight bandage the lungs are put to rest; the general feeling of comfort acting soporifically induces invigorating sleep, the cough generally subsides, chest and pleural pain abate, breathing becomes more free, expectoration easy, sputum less tenacious, more fluid, and general improvement of conditions are noticeable. Tuberculous individuals who are very anemic or undernourished and complain of a cold feeling after this method of hydrotherapy or perhaps manifest rheumatoid or arthritic pains are better treated by substituting warm water for the cold; in fact, in many sanatoria the use of the hot water figure 8 bandage is altogether applied in lieu of the cold. Many patients cannot tolerate a cold dressing at all; besides, the hot bandage also has definite fever reducing tendencies and in patients in whom the cold pack is contraindicated the hot pack may be of decided value. Hydrotherapy is also contraindicated during the menstrual period. In general, however, it may be said that in the treatment of pulmonary tuberculosis, in patients showing much restlessness, nervousness, or much fever, the use of the figure 8 bandage either cold or warm can not be too highly recommended.

"For safety and comfort, then, for freedom from disagreeable symptoms that may or may not be harmful, for certain positive

benefits that cold baths usually fail to give, the hot morning bath is to be commended with great confidence as a daily resource and a daily joy. The cold bath is useful chiefly for those who least need the invigorating effects of any bath, that is, the well and husky people. The hot bath is most valuable for that great mass of our population (especially the tuberculous) who are below their physiologic par and who need every influence that can increase their strength and resisting power. Many of these half invalids and invalids by means of the cold bath are now with true heroism daily subjecting themselves to shivering discomforts often lasting for hours and with no benefit—or worse than none—solely because of a false notion derived from the partial experiences of the strong. It is a shameful waste of courage and power in a good cause. The worst griefs in life are those that are preventable, and the cold bath martyrdom is uncalled for." (Norman Bridge.)

(D) PULMONARY GYMNASTICS
(Pulmonary Massage)

Their Use and Abuse. Their indications and contraindications (173).

Throughout the whole field of conservative tuberculosis therapy individualization becomes the necessary watchword. No two cases can be treated exactly alike; this applies most particularly in the recommended breathing exercises for tuberculosis disease which are known by the simple term—pulmonary gymnastics. The indiscriminate use of these exercises is often followed by the most distressing consequences whilst with proper application in carefully selected cases adequately supervised, the method becomes a most valuable therapeutic measure; hence pulmonary gymnastics or carefully supervised breathing exercises may be very suitable measures if we are only particular in selecting proper cases. For these exercises cases may be considered suitable in which the process becomes arrested, in which there are no active symptoms, beginning cases with little or no activity and a fairly good general condition. Schultzen considers the so-called curable cases, with little or no advanced involvement, no acute symptoms or complications, as the favorable cases, briefly, those cases with increased temperature, rapid pulse, acute pulmonary processes are not suitable for pulmonary gymnastics.

In such cases it is better to keep the range of respiratory motion as small as possible so that there will be no disturbance of the inflamed pulmonary tissue. Two problems must be most carefully considered in recommending these pulmonary exercises; (1) does deep breathing favor or hasten the process of arrest by its beneficial influence on the local and general condition and; (2) is deep breathing, if it produces no observable and favorable influence on the patient, likely to produce harm? We are not all agreed as to its salutary tendency, for instance, Cornet, Liebermeister and others express their opposition to the application of these exercises in pulmonary tuberculosis, as a result of personal experience and upon theoretical grounds. According to these clinicians, there is great danger from aspiration pneumonia, from interference with the formation of a zone of demarkation, a tendency towards hemorrhage, development of emphysema, a tearing apart of the delicate cicatritial tissue and conservative pleural adhesions, conditions, which are favorable to the growth of the tubercle bacillus and increase absorption of toxins, aspiration of bacillary sputum into the still healthy portions of the lungs, etc. This opposition is met by such clinical observers as Dettweiler, Turban, Fraenkel, Schultzen and many others, all of whom have had extensive personal experience; they all favor and employ under carefully guarded conditions pulmonary gymnastics. Dettweiler after giving explicit instructions as to how deep breathing should be applied concludes by saying, "The habit will become a necessity and through its measure the tendency to a troublesome and distressing cough will be blunted," and Turban declares that "deep breathing continuously and methodically employed is most advantageous, especially with the habitually weak breather." He further states that an extreme expansion of a tuberculous lung may result most seriously but that systematic deep breathing increases the ventilation and circulation of the lungs.

Cornet and his school base their opposition to active lung excursion in manifest pulmonary tuberculosis upon the fact that in almost every other form of tuberculosis it becomes essential that the parts are put to rest to favor repair, and if this is true why should not this principle apply to pulmonary tuberculosis as well? So it does. We all, without exception, advocate that in active or progressive pulmonary tuberculous disease the more absolute this rest can be maintained, the greater the amount of

rest which can be put upon the pulmonary tissue the better the prognosis, the quicker the recovery; it is the same principle which we apply in all other forms of tuberculosis. After the process is healed, for instance, after the tuberculous disease in a joint no longer exists do we not all recommend and encourage that passive motion or massage be applied to the joint? Then why should we not advise passive motion, pulmonary massage (and that is all pulmonary gymnastics is) when the tuberculous process in the lungs has become arrested or perhaps healed? After a lung has been compressed for some time, either artifically or naturally, and the pulmonary lesion is supposed to have been healed, do we not recommend pulmonary gymnastics on similar grounds? Schultzen, among others who have had extensive experience in the therapy of pulmonary gymnastics, gives the following contra-indications to their use: (1) in the presence of fever; (2) great bodily weakness; (3) in acute inflammatory processes of the lungs; (4) in progressive softening and disintegration of pulmonary tissue; (5) in increasing extension of the tuberculous process usually accompanied by copious expectorations; (6) in the presence of old or perhaps newly formed large cavities; (7) in recent pleurisies or pleural effusions; (8) in recent hemorrhages; (9) in pathological conditions of the blood; (10) in advanced or ulcerative tuberculous laryngitis; (11) in accompanying intercurrent disease; (12) possible unexpected occurrences such as may be produced or excited by pulmonary gymnastics.

HOW TO PRACTICE PULMONARY GYMNASTICS

Breathing here should be done, as is now so generally taught in our schools, by inbreathing through the nose with the mouth closed and outbreathing through the mouth. Take a full, deep breath very slowly through the nose, the mouth being closed, the breath then being held for a few seconds after which the air is to be exhaled very slowly and fully through the mouth. The act of inspiration can be strengthened by gradually raising both arms to a horizontal position during inbreathing, letting them fall again in outbreathing. The expiratory phase can also be fortified by uttering a few short words at the end of expiration like one, two, three, etc. Usually in the beginning I direct that the patient take half a dozen exercises twice a day, the first thing in the morning on rising and just before retiring. In this way the individual generally becomes accustomed to these exercises so

that gradually he increases the number of deep breathings to one exercise every three hours. A patient with an arrested tuberculous process should take at least six such breathing exercises each day, this to be maintained almost indefinitely. It becomes imperative to caution patients taking pulmonary gymnastics that the exercises must not be vigorous, hurried or forceful but that they must be fairly full but slow, very easy and quiet and that on full inspiration the breath must be held for a few seconds. These exercises may be supplemented by those which I have found most helpful in arrested cases, the patient taking a full breath and while outbreathing carrying the breath slowly along a certain key until all the air is expelled. Begin the exercises at first with a very low key, the next exercise a little higher note and so on until the voice is carried along on the highest possible note. These deep but steady breathing exercises should be taken while the patient is in the upright or standing position; however, for those who are not quite up to full standard, pulmonary gymnastics in the recumbent position may be advised and as the patient improves in general health, deep breathing and other exercises may be judiciously recommended such as singing, loud reading, loud speaking, etc., and in addition to these exercises of full, slow and deep breathing with a proportional resting pause, the cycles of breathing per minute become necessarily lessened in number, perhaps in some instances to nearly one-half giving the tuberculously diseased lung in that proportion a longer period of rest.

"In advising pulmonary gymnastics it cannot be too strongly emphasized that in active tuberculous disease this procedure is proven to do much harm. During the active stage of the disease it is better to produce chest immobilization and not chest excursion, hence this method is advisable only if the tuberculous process is arrested or probably healed and we desire more pulmonary aeration and better blood and lymph flow. From clinical observations by most competent clinicians we can conclude that pulmonary gymnastics carefully applied and supervised both from experience and theory are shown to be useful in aiding, hastening and maintaining the arrest, and that there is no conclusive evidence that any harm results from them. Furthermore the patients themselves express a sense of well-being from such breathing exercises, and besides, the mental effect can not be too highly appreciated." (Dr. E. O. Otis, Boston 1906).

CHAPTER 22

PHYSIOTHERAPY, VOCATIONAL AND OCCUPATIONAL

Rehabilitation—Reconstruction

Historical Data. Hippocrates according to Lunton,(90) (145) wrote upon Airs, Waters, and Places; in the latter part of the 18th Century hydrotherapy was fashionable. The history of work therapy is more obscure. Eva Charlotte Reed states that Galen says "employment is 'Nature's Physician.'" Occupational therapy was advocated in Pinel's writings in 1791. He states that "Moderate employment and regular exercise coöperating with the energies of nature herself, restored him in a short time to the full enjoyment of his intellectual faculties." The translator of Pinel's Traite, Davis, makes reference to much earlier therapeutic uses of occupation and diversion as follows: "At both extremities of ancient Egypt, a country that was at that time exceedingly populous and flourishing, were temples dedicated to Saturn, whither melancholies resorted in great numbers in quest of relief."

Whatever gifts of nature or productions of art were calculated to impress the imagination were there united to the solemnities of a splendid and imposing superstition. The most voluptuous productions of the painter and the statuary were exposed to public view. Groves and gardens surrounded those holy retreats and invited the distracted devotee to refreshing and salubrious exercise. Gaily decorated boats sometimes transported him to breathe amidst rural concerts the purer breezes of the Nile. In short, all his time was taken up by some pleasurable occupation rather than by a system of diversified amusements enhanced and sanctioned by superstition.

Dunton (130) tells us that in 1798 Benjamin Rush, in a letter to the managers of the Pennsylvania Hospital, advocated work as a remedial measure. In 1822 Wyman, Superintendent of McLean Hospital, in his annual report, says that, "The amusements provided for patients, as draughts, chess, backgammon, nine pins,

swinging, sawing wood, gardening, reading, writing, music, etc., have a powerful effect in tranquilizing the mind."

In 1884 Kirkbride stated in an article on the management of hospitals for the insane, "Labor is one of our best remedies. It is useful in improving the health of the insane as in maintaining that of the sane." During the past ten years an unusual interest in the subject of work therapy has developed, in large measure due to the publication of the book by Miss Susan E. Tracy in 1910, entitled "Invalid Occupations." Following the publication of this book it has since been recognized that occupation is one of the best of the means we have for aiding the sick.

In the treatment of pulmonary tuberculosis, Physiotherapy, vocational and occupational, plays a most important rôle. The physical reconstruction of those incapacitated by pulmonary tuberculosis necessitates a long period of continued treatment consistent with the nature of the disability. This treatment may consist in light mental and manual labor, physiotherapy, thermo-electro, hydro and mechanic therapy, massage, calisthenics, gymnastics, heliotherapy and psychotherapy and amusements in and out-of-doors, etc.

Herman Brehmer of Gobersdorf, Germany, first systematically formulated the general therapy for the treatment of pulmonary tuberculosis. He began to do this about sixty years ago, and his methods still exist. He prescribed methodical walking and hill climbing exercises in order to strengthen the heart, the smallness and relative insufficiency of which were in his opinion the chief causes of pulmonary tuberculosis. These exercises were carried out during the day for hours at a time as much as possible in all kinds of weather. They consisted in going slowly up hill or gently ascending paths, in the frequent insertion of cautious, methodical deep breathing and in the avoiding of everything that would tend to tire the patient and of every excitement of the heart.

Brehmer's Sanatorium was built on the lowest spot of a mountain valley, so that all paths that led to it could be so made that there was a gentle down grade.

Brehmer was a great disciplinarian and stated that only one patient among thirteen thousand suffered from over-exertion. This patient disobeyed the rules and over-exerted himself, a severe hemorrhage resulted, from which he died. Brehmer did

not allow patients to do any work with arms and he did not allow them to walk when their temperature was elevated.

Rest treatment is to be ascribed in large degree to Dettweiler. His views, however, were not always fully and correctly interpreted. He was first a patient and later an assistant to Brehmer. At first he employed Brehmer's methods entirely, and in 1873 wrote as follows: "On discussing a broader, very important part of the rational therapy, I mean hill climbing, I must again refer to the circulatory and respiratory, respectively aspiratory anomalies which are to be regarded the necessary presuppositions of subsequent phthisis. From this point of view it was a simple logical sequence to abandon the relaxing regimen of the older therapy, the fear of stimulating the blood vessels, etc., as harmful, and to make use of muscular work to increase the respiratory and circulatory energy."

In Dettweiler's hands, however, some serious results occurred among his patients, and he modified his technic and introduced the reclining treatment. He emphasized most warmly permanent rest treatment in the open air, and warned against too much exercise of the heart and of the lungs. Because of these examples of harmful heart tiring through hill climbing, Dettweiler's rest treatment was carried out in reclining chairs. He built resting halls, open on one side, and placed in them comfortable, adjustable chairs, where most of the patients without fever spent from seven to eleven hours daily. He also employed absolute rest to combat the fever of phthisis.

Edward L. Trudeau of Saranac Lake, New York, may be mentioned as the third great figure in the modern treatment of pulmonary tuberculosis. About the year 1882 he learned of the work of Brehmer and Dettweiler from articles that appeared in "The Practitioner," an English Medical Journal. At first he tried to use Brehmer's methods on his own person. He found, however, that vigorous exercise produced fever and caused a lighting up of his disease, while on the other hand, if he exercised mildly his disease remained quiescent. He soon became convinced that rest in the open air was a more important factor in the cure of pulmonary tuberculosis than exercise, and that exercise was to be employed not in all but only in certain cases.

Inspired by the work of Brehmer and Dettweiler, Trudeau founded the Adirondack Cottage Sanatorium, in February, 1885. It was founded for the purpose of helping the sick, poor and per-

sons with small means to regain their health. His sanatorium has always stood for a rational use of rest and exercise; he never allowed the patients any manual labor. Trudeau's views on the subject of rest are fully described in his letter to Joseph H. Pratt, written in 1911.

Lebert and Rhoden are for abundant bodily exercise at every season of the year, but warn against fatigue. In the last decade of the last century the rest cure was employed in a most thorough manner in the People's Sanatoria in Germany, so that it became known abroad as the German method. Only a few Phthisotherapeutists held strictly to the old regimen of Brehmer: For example, Walther of Nordrach, and Chas. Page, who were strongly in favor of abundant bodily exercise; Liehe and H. Weber, who are not at all antagonists of the reclining method of treatment, consider an abundant amount of exercise indicative for a large number of patients. They are decidedly influenced by Brehmer's school. Liehe is opposed to Penzoldt, who believed that the sparing of the conserving methods is insufficient in sanatoria and who pleads for the most energetic conserving of the strength of the patient on account of the short period of time of the institutional treatment and speaking warmly in favor of systematic exercise therapy. In the epoch in which the conserving or saving therapy was practiced in an exaggerated manner, there were always clinicians and phthisotherapeutists of repute who favored systematic exercise treatment on the ground of strict indications. Such clinicians were Cornet, Meissen, Frankel, Turban, Penzoldt, Trudeau, Vincent Bowditch, and E. R. Baldwin and others.

Rational Physiotherapy

Today it has become a principle of rational treatment to carry out sensible, methodical exercise treatment. We can also determine the indications and forms of employment so that exaggerations toward the one or the other side can be avoided, taking for granted, of course, that the physician is able to judge of the lung condition. Physicians at times differ as to the amount of exercise considered proper for patients in tuberculosis sanatoria. The more radical have advocated laborious work, such as building, masonry and ditch digging, while at the other extreme are those who are strong advocates of the rest treatment, with the addition of calisthenics and walking exercises.

Recognizing physical exercise as a therapeutic measure patients at certain sanatoria are expected to perform a sufficient amount of work to insure physiological reaction on the part of the various tissues (skin, glands, muscles, etc.), which if left dormant for a considerable time would, through their failure to functionate, retard the patient's recovery.

Work assignment in a sanatorium should be a therapeutic measure and secondary to the medical care of the patients. Work assignment is an expedient used to divert the thoughts of the patient from his physical condition to other things. It may be expressed in the terms already mentioned, vocational therapy, graduate exercise, rehabilitation, occupational therapy, reconstruction, vocational education, vocational training, etc.

a. Rehabilitation of the Handicapped. The Tuberculous Soldier

While there is a direct relationship between occupational therapy and the war, many similar problems are also met with in civil life. We have industrial cripples from other causes, and these should be aided in becoming at least partially self-supporting, and should be taught some occupation which will help them to pass their leisure time pleasantly if not profitably. It is of interest to note that the great war has done much to emphasize the value of occupational therapy.

In the first place the crippled have been taught methods by which their handicaps may be overcome or discounted, and secondly, occupational therapy has proved extremely valuable in the treatment of those functional disorders to which the name shell shock has been given.

It became evident soon after the war began that the number of crippled returning to civil life would be so large, that the old plan of pensioning them would be impossible to carry out, especially as the number of able bodied would be too few to support the tremendous financial load of taxation which would be necessary. The problem being an economic one attracted more immediate attention abroad than if it had been merely medical, and for this reason M. Herriott of Lyons, France, established two schools where the crippled were taught new trades or could learn to adapt themselves to former ones. The physical reconstruction of soldiers in this country, disabled by pulmonary tuberculosis during the great war, was a new problem. Occupational therapy and vocational training were recognized by the Federal Board

(138) for Vocational Education as most valuable as an auxiliary means of treatment in tuberculosis. As applied to the treatment of soldiers in this country, it has proven an emphatic success, and especially so in a disciplinary and moral direction. Instruction has been provided in general subjects, commercial branches and light arts and crafts work, and, where possible, gardening and greenhouse work as well. It is designed that all training shall have a definite vocational bearing so that upon discharge, if the patient does not wish or is unable to undertake his former work, the experience which he has gained during his stay in the sanatorium may help him directly in earning his living.

It must be understood that there are many problems covering this subject yet to be solved, and it is hoped they may attract the attention of the research worker. There are many difficulties to be encountered concerning the emotional reaction of the patient. Men of the same mental caliber and from the same social level will differ in their adaptability to a given occupation one form of work appealing to one man and not to another.

We have acquired increasing knowledge as to the value of work as a remedy and occupational therapy applied along the lines of reconstruction will give added value to this important subject. C. L. W.

b. Reconstruction of the Tuberculous in Civil Life

The most essential treatment of the tuberculous individual during and after sanatorium care is by regulated[1] "rest and exercise." (70) The principles of sanatorium treatment are so definitely standardized today as are those of surgery, but they are not nearly as familiar to the profession as is surgery. In every case of pulmonary tuberculosis, we must individualize in the application of these two most essential principles of rest and exercise. While one patient should not be permitted to raise his hand, where absolute rest is required, another may do a heavy day's labor; between these extremes every case of tuber-

[1]The tuberculous individual suffering from active disease is first put to rest in order to lessen the amount of tissue change, slow the pulse and reduce the temperature. This lessens the amount of autointoxication, autotherapy and autotuberculin absorption. If the process has been arrested and the patient's condition has improved he may be allowed a little exercise just sufficient to keep his autotherapy within normal limits; if, however, he exceeds this and the exercise in whatever form it may be, is excessive then the autoabsorption of tuberculin becomes too great, his therapy too high and the conditions are again bad. The active tuberculous individual at all times has sufficient tuberculin within his organism for normal medication and use, hence it must be our aim to keep his therapy within these limits so that the prescribed exercise pours just sufficent autotuberculin into the economy to act as a stimulant to the production of antigens and antibodies and the amount of labor demanded must be accurately graded for each individual case as best will bring about a rational autotherapy.—J. R.

culosis can be placed. It becomes necessary to know how and when to direct, as fixed rules can not be applied. A good rule is not to put the arrested tuberculous at once to physically hard labor, but with returning good health heavier work may gradually be added, and it should be remembered that it matters very little whether this labor is performed in or out-of-doors so long as the vocation is healthful and not too strenuous. It is a very serious mistake to impress upon the tuberculous' mind that on his return from the sanatorium where the disease has become arrested, he must follow a vocation different from the one he previously pursued. As the doctor must be guarded, must weigh carefully, in recommending a suitable sanatorium, in advising a change of climate, a sojourn in the mountains or at the sea-shore, so must he be equally guarded in recommending a certain vocation to follow for a livelihood. A returned tuberculous individual whose pulmonary process has become arrested will do best to follow the usual trade or calling which he followed for a livelihood before he entered the sanatorium, perhaps in a modified form, something light, but related to his previous training. For example, a machinist may learn to become a draftsman; a carpenter, an architect; a bookkeeper, a secretary; a doctor, a laboratory diagnostician, etc. Put the healed or arrested tuberculous to work at a vocation or trade as near as possible to the one with which he is familiar. The learning of a new trade entails a hardship and is more of a handicap than an asset, and above all the patient will look with longing eyes to the work with which he is acquainted. After years of training along certain lines, it is quite hard to take up new duties, especially so, if the family is dependent upon such an undertaking. Strictly regulate the hours of rest after work, regulate the hours of work, the individual's living, sleeping, resting and eating conditions, and much can be accomplished. Entering into a new field is most difficult. It is better to become more efficient in the old and familiar one.

Do not recommend that the tuberculous patient become a farmer, to raise cattle, sheep, horses, or poultry, to take up gardening or forestry, to become a traveling salesman, or become a timekeeper and to stay in the open in all kinds of weather. To follow any of these with success, to make a living, or perhaps take care of a family, requires work, and often times the hardest kind. It is hard because he is not sufficiently familiar with that

kind of work, and as he had pictured these vocations as easy to learn, he becomes disheartened when he finds himself deceived.

Reconstruction therapy then, for the arrested tuberculous in civil life should be supplied as near as possible to the individual's interests, his likes, desires and aptitude and should be especially related to his former field of usefulness. Most tuberculous individuals returning from the sanatorium with their pulmonary disorder arrested have families or dependents, and they are very anxious to again become efficient working units in the community. The learning of a new trade, to master it so as to meet all competition, usually requires years of training, and can not be acquired in a short time; hence the suggestion that the healed tuberculous remain in the calling related to the one with which he is fully acquainted or follow the old vocation modified for easier labor, is the only good, rational and logical one. This is efficient reconstruction therapy. J. R.

"Graduated Labor"—"Rest and Exercise"

Paterson's Method for Applying "Exercise and Rest" Treatment.(171) Paterson's work was carried out systematically in the Brompton Hospital Sanatorium at Frimley, England, where patients were directly in his charge over a number of years, and it was largely through his work that "graduated labor" as an important method of treatment gained more or less popularity in England and America. All patients were carefully selected and only fever-free patients and those free from other constitutional symptoms, and who had begun to show some signs of improvement were chosen. Several thousand selected cases were treated by this "graduated labor" method, and many of these patients had been able to perform five hours of hard labor each day without any apparent ill effect before leaving the sanatorium. It is shown that after returning to work, at the end of the first year but 62% were well and working, and this was lowered to 50% at the end of the second year.

According to Paterson, the treatment by rest and exercise is a logical deduction from the theory of auto-inoculation,[2] and auto-inoculation is descriptive of a process which consists in the in-

[2]Auto-inoculation in every respect simulates tuberculin therapy. With little exercise, little tuberculin stimulation go hand in hand for the better, whereas with much exercise like much tuberculin, much damage, and even dangerous conditions may arise. Auto-inoculation is auto-tuberculinization. The judicious use is highly beneficial whilst the injudicious use is extremely dangerous.—J. R.

troduction of bacterial products into the blood. As Wright states: "All active and passive movements which affect the focus of infection, and all vascular changes which activate the lymph stream in such a focus induce auto-inoculation by means of which the blood begins to provide for itself a protective mechanism." The process is one of immunization, and as a consequence, whenever bacterial products escape from the localized foci, intoxication phenomena and immunizing responses must necessarily supervene.

Where the auto-inoculations continue and the immunization process is weak, death is inevitably the end. Where the defense is progressively equal to the attack, the assaults of the bacterial products are gradually worn down until the rising levels of resistance produce in the end complete immunity.

Paterson calls attention to the fact that treatment by means of exercise is not of general application; it can only be used in the case of a patient who fulfills two rather onerous conditions.

First he must be afebrile and free from all constitutional symptoms; and secondly he must have attained the position of an ordinary person in the house by being able to remain up all day fully dressed, and to walk up and down stairs.

The following classification of patients is made:

1. Those who have been infected and have recovered without knowing of their infection.
2. Those whose recovery is certain after rest and a holiday.
3. Those who require additional help to that conferred by general hygiene.
4. Those for whom no treatment will do any good.

Therefore, there are only Classes 2 and 3 where treatment by the type under consideration is possible. These, again, are divided into febrile patients and patients free from fever.

Paterson states that the control of excessive auto-inoculation by absolute rest is complimentary to its artificial inducement by exercise and must be instantly adopted upon the slightest return of fever.

Fever means a temperature exceeding 99° F. in men and 99.6° F. in women, accompanied by constitutional symptoms, such as malaise, headache, and similar signs of hyper-intoxication. A temperature of 100° F. in a case where there are extreme signs in the lungs can be ignored, provided that there is absence of all constitutional disturbance.

The immobilization of patients must be absolutely effective, and in addition to bed rest, patients are not allowed to move or to read; talking is forbidden, and steps are taken to repress any needless coughing, if necessary, by wearing a Burney Yeho's inhaler. The use of the stethoscope is debarred, since it entails the taking of deep breaths. Visitors should be kept at a distance, and even the patient's letters should be withheld if there is any probability of the intrusion of home worries. Six to ten days of such regime are in most cases sufficient to bring the patient's temperature to normal. If the temperature remains normal, the patient is allowed to sit up in bed for half an hour, and the period is gradually extended until the patient is able to dress and remain up throughout the day.

Should prolonged trial of immobilization fail to reduce the fever, more complete immobilization may be considered by the introduction of nitrogen gas or filtered air into the pleural cavity to neutralize the intrapleural negative pressure.

Graduated exercise and systematic labor mark the second control. Patients who have been immobilized, owing to the presence of fever and constitutional symptoms, begin by walking 440 yards a day. The temperature and constitutional symptoms form the guide throughout, and should there be no marked deviation, this exercise is continued for a week, and is then increased to 880 yards per day. The third week two miles is prescribed, which is increased to four in the last week of the month, and the final walking stage is over six miles, and is attained automatically.

Cavities involving four lobes with good muscular condition form no reason for the proscription of the quarter mile grade. Such a patient can be started on the four or six-mile grade, whereas the patient having little or no physical signs, but with poor physique, must be watched carefully. With the revival of no unfavorable symptoms, the grades of labor are entered upon successively.

Grade I consists in carrying for a distance of 50 yards up a gradient of 1 inch, 1 to 7 baskets containing materials and averaging about 10 pounds in weight. The number of times daily should be adjusted to the distance of seven miles.

Grade II is a slight extension of Grade I, 18 pounds of material are carried a longer distance in a larger basket; while the third grade allows of more arduous work, such as chopping fire wood, cutting grass, hoeing, etc. The fourth grade is shovel

work, or its equivalent: mowing grass, sawing trees, etc. Two tons of earth lifted into a cart is the daily task in the beginning, gradually increasing to above twice that amount. In Grade V six tons a day is the prescribed amount, a larger shovel being used, while patients in the last grade are required to do five and a half hours of the heaviest type of work.

The average time for the various grades is as follows: Grade I, one week; Grade II, one or two weeks; Grade III, one or two weeks; Grade IV, two weeks; Grade V, three weeks, and the patient is then permitted three weeks from his discharge, to work on the heaviest grade.

Paterson calls attention to the fact that an important aspect of a system of sanatorium treatment is the demoralization of the sick, produced by the mental and physical lassitude of long periods of inactivity. Graduated labor from this point of view has a psychological and moral value impossible to over-emphasize. C. L. W.

CHAPTER 23

TUBERCULOSIS IN CHILDREN

Tuberculosis of the Tracheo-Bronchial Lymph Nodes.(15)

Here especially we must recognize the difference between being tuberculously infected and tuberculously diseased. The tracheo-bronchial lymph nodes being regional glands are usually secondarily infected after the primary infection, but tuberculous disease of these glands is primary tuberculosis.(183)

In children tuberculosis is primarily localized in the bronchial glands, and the clinical picture of tuberculosis so often seen is that of an extension of the disease from these glands to other organs of the body, to the meninges, the lungs, the bones and joints, the kidneys, spleen, etc. After puberty, pulmonary tuberculosis by far displaces all these various forms.

In very early infant life the tuberculous process is usually rapid, the glands having not yet acquired the power of resistance to the disease and consequently rapid dissemination follows, the disease usually running a rapidly fatal course, but after infancy and from child life to puberty the tendency to a generalized dissemination is lessened, the glands having acquired a greater degree of resistance, and now a localized gland tuberculosis, chiefly of the bronchial glands, is the clinical picture presented. However, at this period, intercurrent conditions may arise, such as injury and shock, measles, whooping cough, etc., which may again lower the resistance of these bronchial glands, and material being disseminated may again be followed by generalized lesions, miliary tuberculosis, meningeal, bone and joint disease, pulmonary tuberculosis, etc.

Enlarged bronchial glands do not always lead to active disease, on the contrary in the greater number of instances the conditions terminate favorably, the glands undergoing calcification and becoming encapsulated, but if the contents should undergo softening, and the soft material enter the lymph channels or more particularly the blood vessels, then this would be followed by speedy miliary disease. This latter may occur at any time during

infant and child life even into adolescence and up to old age. Tuberculosis in child life often manifests a great degree of latency, and frequently after a period of apparently perfect health, suddenly, as though out of a clear sky, an attack of tuberculous meningitis may appear, after a very short and stormy course leading to speedy death from miliary tuberculosis.

Clinical tuberculosis, as has been previously stated, may be divided into three stages. After infection has taken place, the regional glands become secondarily involved. This may be followed by primary tuberculosis of these glands, and the process may terminate here, or it may extend at any time to the second

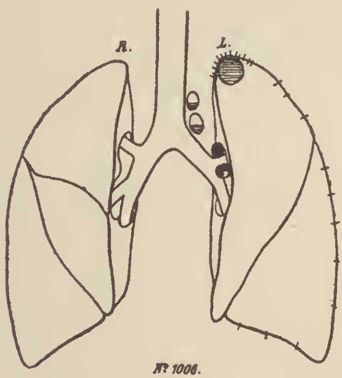

Fig. 41. The Lung (schematic) after Ghon. Autopsy No. 1006. Cause of death generalized Tuberculosis, male child, 3 years. Note the cavity in the apex of the upper left lobe surrounded by an adherent apical pleuritis, and pleuritis of the greater portion of the left pleura. The regional lymph nodes, the left broncho-pulmonary have undergone complete caseation and partial caseation of the tracheo-bronchial.

stage by metastatic involvement of other organs or tissues, either hematogenous or lymphogenous, or it may manifest itself in years after as chronic pulmonary, the third or tertiary stage of tuberculosis. Tuberculosis in infants and children is usually primary or secondary, and only exceptionally tertiary.

(A) The Primary Stage of Tuberculous Disease in Children.(77) Primary Tuberculosis. Clinical Manifestations as Seen in Infant Life.

In children somewhat advanced in life definite symptoms are generally absent. In small children, however, the clinical pictures are those of fever, emaciation, anemia, etc. The fever may have the typical every day rise. Emaciation may come on suddenly or be more or less protracted. Night sweats are more fre-

quent in the elder than in the infant life. All these signs are very indefinite and although suspicious of tuberculosis, may have to be confirmed by the tuberculin test.

Bronchial Gland Tuberculosis: Tuberculosis of the Tracheo-Bronchial Lymph Nodes. Glandulae Tracheo-Bronchialis.

The lymph flow from the upper portion of the lungs is to the upper group the tracheo-bronchial glands, the middle and lower to the lower glands, and these glands communicate with the deep mediastinal and with the glands of the supraclavicular region about the bulbus vena jugularis, and not with the cervical glands, nor with the tonsillar lymph nodes; hence in speaking about gland tuberculosis only the tracheo-bronchial lymph nodes, and perhaps the deep-seated mediastinal, are considered, and not the palpable cervical glands. Bronchial or more correctly peri-bronchial gland tuberculosis is most frequently found in children, and only occasionally and exceptionally in adults. Primary gland tuberculosis is as infrequent in adults as is pulmonary tuberculosis in children. The cause of the disease in these organs is, like tuberculosis anywhere in the body, the tubercle bacillus, either the human or the bovine. In bronchial gland tuberculosis the human bacillus is most frequently the causative factor, while in tuberculosis of other regional glands the bovine bacillus may play the more important role.

Infection with the human bacillus usually takes place after birth, exceptionally before; (see Chapter 3) and whereas a single mild infection may often give immunity against future infection, a massive or a frequently repeated small infection may be followed by early manifest disease and death. The period of infection may be at any time after birth and up to about the sixth year and infection after that time is very infrequently followed by active disease. Tuberculosis may rightly be compared with lues, that is, after infection, a period of incubation follows, lasting a short time, then a more or less protracted primary manifestation of the disease occurs, and later secondary, and still later tertiary symptoms. In children the primary manifestation of the disease is usually at the cradle, that is, before the school age, the secondary or metastatic form at the school age, and the tertiary, the pulmonary from after the school age or at the high school years, that is, at puberty and beyond. Nearly all pulmonary tuberculosis and which is the tertiary form of the disease has been

preceded by the glandular or primary form. Bronchial gland tuberculosis is primary tuberculosis, may manifest itself at any time after infection, or it may become more or less subacute, and later in child life assume the chronic form.

The anatomical seat of primary tuberculosis is usually in the regional glands or glands nearest to the first or primary infection, and as in the human body, this primary infection is either subpleural or parenchymatous, that is, in the lungs, the glands nearest to the root of the lungs, the tracheo-bronchial, are secondarily infected, and it is here that primary tuberculous disease manifests itself. Three groups of glands are generally involved, those along the trachea, those at the bifurcation, and those at the hilum, communicating with the deep mediastinal. The palpable cervical glands, if tuberculous, are not of this origin, but are due to infection from the mouth, teeth, tonsils, etc.; this enlargement may also be from various other microörganisms.

Signs and Symptoms of Gland Tuberculosis

The symptoms may be very characteristic in child life, or there may be absence of all objective signs, and again in many instances there are no definite signs, with perhaps the single sign of pain. Pain in the chest anteriorly, mainly to the right of the sternum and posterior interscapular backache, is much complained of. A painful cough with an expiratory stridor, however, is usually present, due to the swollen glands pressing upon the bronchi, producing pressure symptoms and there may be a metallic cough and expiratory dyspnea, whooping cough-like symptoms supposed to be due to irritation of the branches of the vagus. This cough, hollow, barking, metallic, sounds like a whooping cough and is differentiated only by close study and long observation. In ordinary whooping cough there is usually an inspiratory dyspnea, with attacks of vomiting and cyanosis; in bronchial gland tuberculosis the attacks are generally accompanied by an expiratory dyspnea. This piping and particularly high expiratory murmur is much increased after exertion or excitement. It is more protracted in the very young than in the elder children. Other signs occasionally observed are emaciation without any definite assignable cause, nutritional and gastric disturbance, discharges from ears, eczematous patches about the ears or multiple abscesses, slight fever at first, and bronchopneumonic areas at the base of the lungs. In some instances the

swollen bronchial glands may cause pain in swallowing, or there may be loss of voice from pressure upon the recurrent laryngeal nerve, and on examination of the trachea by means of the endoscopic mirror, encroachment of the lumen and bulging of the tracheal wall may be revealed. Pressure upon blood vessels often gives distinctive signs. If the superior vena cava is compressed the vessels of the chest and neck become more full, the face becoming eventually edematous and cyanotic. If the veins of the lungs are compressed, a distinct tendency to nasal bleeding often results, and by the Valsalva method the veins of the neck over the affected side may become congested and pressure upon the arteria pulmonalis may produce a systolic murmur. In a healthy child, if on deep inspiration a Valsalva is practiced, the pupils become contracted, while in the diseased the pupils will become dilated, especially on the involved side.

Pathologic-anatomically the right bronchus is most frequently compressed from enlarged and caseating glands, mainly about the bifurcation, and bending of the bronchus towards the upper lobe, the expiratory dyspnea, a harsh loud sound, expiration much prolonged, inspiration scarcely audible, accompanied by inspiratory contraction of the chest due to the pressure of enlarged glands upon the trachea, if continued for some time, and if accompanied by emphysema of the lung, the face becoming congested, the veins dilated, cyanosis, and in protracted cases clubbing of the fingers, all bespeak primary or bronchial gland tuberculosis. Physical diagnosis is usually negative. In spite of the frequency of bronchial gland tuberculosis in the first and second and perhaps up to the fourth year of child life, these characteristic signs are but seldom observed, and endothoracic gland enlargement is during life infrequently diagnosed.

Physical Examination and Diagnosis. As in the examination of the chest in pulmonary tuberculosis, so we apply the classic methods of physical examination by inspection, palpation, percussion, auscultation and roentgenology for the interpretation of the signs of bronchial gland disease.

Inspection usually presents a frail child, chest somewhat contracted, Adam's apple more or less prominent, and in many cases the pupil on the affected side is dilated due to pressure upon the sympatheticus by enlarged swollen glands; there may be unilateral sweating or flushing of the face; the temporal veins on the affected side are usually prominent and enlarged; the skin is

usually dry and covered with a downy growth of hair.

Palpation. Interscapular backache is frequently complained of. If the physician places the tips of the fingers of one hand along the spine, and the other hand over the sternum and slight pressure is made from above down and again up, the child being placed sideways, standing before the physician; and if then close attention is paid to the child's facial expression while this is being done, one will often observe that when certain points along the spine are touched, the child shows signs of flinching as if in pain. This technic was first described in detail by Petruschky under the term "spinalgia." Pressing upon the thoracic spines from the second to the seventh, particularly on the third and fourth, will often cause pain. In palpating the upper intercostal spaces anterior, near the sternum, towards the right side pain is also often elicited if the glands are swollen. It is to be borne in mind as a differential diagnostic point that often in cardiac disease, pain on pressure can frequently be elicited over the first to fourth dorsal spine and in stomach disorders from the fourth to the eighth.

Percussion. Percussion anteriorly over the sternum is of little value, as an enlarged thymus or a deep and low lobe of the thyroid may give a dull note and a dull note along the parasternal line right side between the second and third interspace is usually not due to enlarged glands, but to a retraction of the right lung border. Korányi has pointed out that posteriorly in the normal condition, the percussion note may be dull and osteal over the spine to the fourth thoracic vertebrae, and pulmonary below that point, but that dulness to the fifth and sixth dorsal spine is always pathological. This dulness may extend on both sides or to either side to the paravertebral line. With the dull note the resistance is also increased.

Auscultation. Endothoracic gland enlargement can often be diagnosed by means of the stethoscope. If listening with the stethoscope along the spine of a normal child while breathing, beginning in the neck up over the fourth or fifth cervical vertebra, we will note distinct bronchial breathing, and as we proceed downward the sound will continue, with perhaps a little less intensity but of the same quality until we pass the first thoracic spine, when it becomes fainter and is nearly lost at the second. If next we listen along the spinous processes of a child whose bronchial glands are enlarged, the tubular sound which we hear

up in the neck will be carried downward, often as far as the sixth and seventh dorsal spines and beyond (Spino-trachael breathing). D'Espine has pointed out that if we listen with the stethoscope along the spine from above down and slightly to either side while the patient counts one, two, three in a whisper, the whispering voice in the presence of enlarged endothoracic glands can often be distinctly heard as far down as the seventh thoracic vertebrae. This bronchophonic voice is usually accompanied by a distinct echo, that is, as the patient counts one, two, three, an additional but slightly fainter three is heard. In normal children the whispering voice sounds are not heard or perhaps very faintly below the first or second dorsal.

The humming sound occasionally heard when listening over the anterior chest along the clavicles and sternum while the head of the child is drawn upwards and backwards, and which is usually known as Smith's sign, is not of much diagnostic value. This sound is very often heard in children up to the fourth and fifth year and occasionally beyond but is not common after the twelfth. It is supposed to be due to the pressure induced upon the innominate vein by the enlarged endothoracic glands while the child's head is extended.

O, de La Camp considers the physical possibility of diagnosing bronchial gland tuberculosis chiefly the tracheo-bronchial situated at the hilum. The symptoms are variable and are chiefly present in the pronounced cases although the principle symptoms of gland enlargement are usually always present. The characteristic dry, brassy cough, slightly husky voice, difference in the pupils, pain between the shoulder blades, spinalgia (Petruschky) pain on pressure from the second to the seventh dorsal spine, and the passing of a sound into the oesophagus causing pain (Neisser), etc. Percussion over and near the sternum anteriorly and posteriorly in the paravertebral line to the right and left of the spine is of less value than percussion directly over the spinous processes as was first demonstrated by Korányi, a relative dulness over the fifth and sixth dorsal spine by moderate percussion indicates gland enlargement.

Tuberculosis of the endothoracic glands in a child is the first stage of the disease. Its early recognition and treatment is of the greatest moment and we are without doubt in a position early to recognize bronchial gland disease and by means of proper therapy at our command we can successfully treat it. Tuberculin injections and radiographic examinations are only auxiliary methods in the hands of a capable and competent clinician.(89)

Roentgenology. The roentgenology of tuberculosis in children. Peribronchial. Pulmonary. Miliary. Differentiation. (See Chapter 17.)

An X-ray plate is the objective representation of that which can be seen by the Roentgen ray. A radiographic examination is often of great aid, and may lead to an immediate diagnosis. It is especially valuable in children, where the clinical examination is difficult and its findings often misleading. Technically, instantaneous exposures are essential on account of the rapid respiration and restlessness of children.

Solitary or multiple (rarely more than four, usually all on one lobe) pea to bean sized, dense sharply circumscribed shadows, which lie free in an otherwise clear lung, may represent, in young children, a primary tuberculous focus (Ghon). If so, definite shadows of the enlarged, regional or corresponding peribronchial glands are usually present, and the v. Pirquet reaction is positive. An active, upper lobe, tuberculous process, associated with a positive tuberculin reaction, appears in children in two types of shadows.

1. Fine, flaky or streak-like shadows in one or both upper lobes, which intensify the normal markings, are heavier near the hilum, and radiate outward, are suspicious for tuberculosis in young children. Peribronchial gland shadows are usually present. The radiating shadows may or may not extend to the periphery. Percussion and auscultation usually reveal only prolonged and harsh expiration. A positive v. Pirquet in these cases makes the diagnosis probable.

2. In the more advanced form, the shadows are not only more extensive, but the flakes are larger and denser. The streaks run into one another, and although appearing to coalesce, still show that they are individual shadows. There is frequently a high position of the diaphram on the affected side, with diminished respiratory movement. Clinically, there is dulness, harsh, often bronchial breathing, cough, and tubercle bacilli may be in the sputum.

On plates this shadow is to be differentiated from that given by any chronic inflammatory process, such as asthma or chronic bronchitis. These latter shadows are evenly distributed over both lungs, and extend well toward the periphery. They are well defined and linear. Sometimes the markings on one side are a little heavier than on the other.

In older children, with advanced tuberculosis, shadows are usually found in the upper lobe of one or both lungs. These shadows are cloudlike, composed of various sized, smaller or

larger, individual shadows which have become confluent. These never become homogeneous in density, as found in pleuritis, lobar pneumonia, or exudate. Frequently there are present well defined clear areas, or a few small well defined shadows, the latter in the vicinity of the larger, of the confluent shadows. The lower parts of the lungs are usually exceptionally clear.

In other cases a triangular shadow may occur in the middle part of either lung. The base of the triangle is toward the central shadow; the apex extends toward the periphery, diminishing in density as it extends outward. In rare instances the position of the triangle is reversed. One margin is often sharply defined along an interlobar septum, while the other, usually the upper, is irregular and not well defined. Again, both margins may be ill-defined. The rest of the lung is clear. Such a shadow speaks for lobar pneumonia, either early or after the crisis, but might be intrapulmonary tuberculosis. The latter is differentiated from pneumonia, in that it does not change, or but slowly, on repeated examination. If it is tuberculosis, several small, isolated, sharply circumscribed, dense shadows are scattered through the area involved. The corresponding glands in the hilum give heavy shadows. Upper lobes are often involved. In children with these findings there is persistent fever, cough and emaciation, with a positive tuberculin reaction. The shadows seen in subacute tuberculous processes in children may be general or localized.

Sharply circumscribed shadows, usually small, but not all of even size, which lie free in the lung fields and are irregularly distributed over one or several lobes, speak for disseminated, caseated or calcified, tuberculous foci, in children with a positive tuberculin reaction similar shadows in the apex, which do not communicate with the hilum, speak for a healed apex tuberculosis, in the presence of a positive tuberculin reaction.

Diffuse cloudiness of the apex region, associated with a positive tuberculin reaction, and with pronounced dulness without change in breath sounds, speaks for a healed tuberculous pleuritis. There may be a narrowing of the apex, or a shrinking of the whole side, due to the pleuritis, to the stenosis of a bronchus, or, more especially, to pressure from tuberculous bronchial glands. Diffuse cloudiness of the apices can also be caused by scoliosis, heavy muscles, struma, supra or infra clavicular glands or cervical rib, etc.

The general type of lung tuberculosis in children extends from the hilum toward the periphery. Primary involvement of the apex, either with clouding, or with more definite markings, is rare in children, almost never occurring before the sixth or seventh year.

Roentgenologially, increased density in the region of the hilum, is of more significance in children than in adults. This appears, on X-ray plates, as rounded shadows, often as heavy as the heart shadow. These may be single or multiple, large or small, in masses or discrete. The margins are usually fairly smooth. They may be hazy and indistinct. Such shadows may be covered by the heart and not be visible, or only dimly made out. These shadows correspond to peritracheal and peribronchial lymph gland involvement, and if associated with a positive tuberculin reaction, speak for tuberculosis.

Acute hematogenous, or miliary tuberculosis may be definitely diagnosed from X-ray plates. In children it appears in two types.

First, that consisting of numerous, nearly equal sized shadows, (possibly larger in the upper lobes), evenly and symmetrically distributed over both lungs. These shadows are just visible in size and lie very close together simulating a cloudy veil which may even blur the central shadows. In the second type they are very similar, but larger and farther apart.

Extra-pulmonary lesions, such as thickened pleura and hydro and pneumothorax are similar in children and adults. (See Chapter 17.)

A tuberculous abscess is a dense, rounded sharply defined shadow, lying near the spine. Its level corresponds to the vertebra affected, or depends upon how far down the cold abscess has descended. C. B. R.

Tuberculin. For diagnostic purposes in suspected gland tuberculosis, the use of tuberculin finds its greatest application. The use of tuberculin as a probationary and therapeutic remedy is fully described in Chapter 24, "Tuberculin and Its Uses."

(B) The Secondary Stage of Tuberculous Disease in Children. Secondary Tuberculosis. The Secondary Form of the Disease.

Dissemination of tubercle bacilli through various channels, a metastatic form of the disease. In many there is no picture of activity. It does not appear in children infected in later life, that

is in those infected after the sixth year and it is usually the cause of death of all those children infected in early life and who have not succumbed to the primary infection.

Two forms are generally recognized:

1. A very malignant form. This form of tuberculosis manifests itself either as an acute miliary tuberculosis or as a basal meningeal. This form is usually hematogenous, the tubercle bacilli gaining entrance directly into the blood stream from a caseating or degenerating gland.

2. A more or less benign form, appearing usually somewhat later in infant or child life in the form of bone and joint tuberculosis, chronic meningeal or spinal tuberculosis, etc. This form is most likely lymphogenous. Many bacilli after entering the lymph channels are held back by the nearest glands and only few gain access to the circulation.

(1) a. Acute Miliary Tuberculosis in Children

The most malignant form of the secondary stage is the acute miliary, a hematogenous infection. In children between two and five the meningeal form is the most fatal. If the brain is not

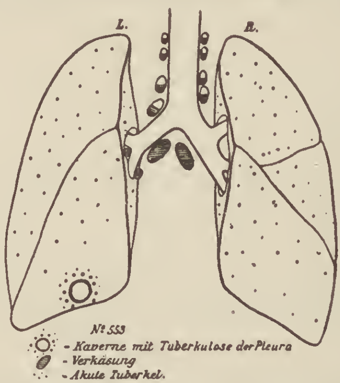

Fig. 42. The Lung (schematic) after Ghon. (Posterior view.) Autopsy No. 553. Cause of death tuberculous meningitis, male child, 5 months. Note the large cavity in the lower part of the left lower lobe surrounded by a tuberculous pleuritis. The regional lymph nodes the left broncho-pulmonary and the epi-bronchial have undergone total caseation, and partial caseation of the upper tracheo-bronchial and para-tracheal on both sides. Miliary tubercules in both lungs. The initial primary and older lesion was in lower portion of the left lower lobe.

swamped with many bacilli then the symptoms of a generalized miliary form prevail. We notice here again a period of quiescence, and from the spread of the bacilli until beginning of manifest disease about two weeks elapse usually without prodromal symptoms. These miliary symptoms last from one to three

weeks, seldom longer. An increasing bronchitis may reach a high stage with severe cyanosis, mind being somewhat impaired, with signs of meningeal disease. Glands may suppurate and perforation into a bronchus or into the oesophagus may take place, usually toward the point of least resistance, or the contents of the glands may gain access into the lymph channels and in that way produce a secondary miliary tuberculosis.

The affection may manifest itself at any age and at any time, usually under the form of an acute infection, beginning either suddenly or with pulmonary symptoms, loss of appetite, slight feverishness, indifference and drowsy condition of one or two weeks' duration. This is quickly followed by a rise in temperature, an irregular pulse, negative lung findings, dyspnea, cyanosis, a dry hacky cough, with expiratory stridor, little or no expectoration, enlarged spleen, and a strong tendency to meningeal symptoms. The diagnosis often offers many difficulties and is largely dependent upon the tuberculin test, examination of the lumbar fluid, and the ophthalmoscopic examination (chorioidal tuberculosis). It may be difficult to differentiate from typhoid, although it usually is by the negative Widal, from lobar pneumonia, bronchopneumonia and capillary bronchitis by the pulmonary findings, and even a localized bronchial gland tuberculosis may simulate the acute miliary form. The course is usually short, fast and very stormy, and exitus letalis from a few days to a few weeks.

(1) b. Basal Meningeal Tuberculosis in Children. Tuberculous Meningitis.

Acute basal tuberculosis in infancy is less infrequent than we are forced to believe. It may often accompany an existing otitis media or mastoiditis, or a previous cerebral tuberculosis. The infection is usually hematogenous from a caseating tracheobronchial gland, the bacilli being carried into the meshes of the arachnoid at the base of the brain. The diagnosis may often be made with great difficulty, owing to the symptoms simulating tetany. The diagnosis must be based on the clinical findings, these very often being meager. Lumbar puncture may be resorted to. The prognosis is grave. Therapeutic remedies are useless. Pain may be relieved by sodium bromide. Morphine is indicated in sufficient doses.

(2) A more or less benign form. A lymphogenous infection. The second form of secondary tuberculous disease. A metastatic

form of tuberculosis. This form of tuberculous disease so frequently seen in children during the school age is fully described in Chapter 30, "Tuberculosis of Bones and Joints."

(C) The Tertiary Stage of Tuberculous Disease in Children.

Tertiary Tuberculosis. The Tertiary or Pulmonary Form of the Disease.

This form of tuberculosis is the usual pulmonary form which we see so persistently throughout adult life but infrequently in children; however, in children, it usually runs a much faster course, and as it does not differ from the tertiary form of the disease in the adult or from pulmonary tuberculosis, as mentioned in the text on pulmonary disease, the definitions given there are applicable to both and a review of Chapters 10 to 18 inclusive is advised.

Therapy.

The Treatment of Tuberculosis of the Tracheo-bronchial Lymph Nodes.

(1) Prophylaxis: To prevent infection of the child and to increase its resistance.

It becomes the duty of the mother, if possible, to protect her child from infection, after infection it becomes her duty to prevent the child from becoming tuberculously diseased, that is, to increase its resistance and here good housing conditions, wholesome fresh air, out-of-door living, with frequent baths, good food, etc., are the necessary factors. The well known axiom of v. Behring, "Tuberculous disease in the adult is simply the last tune of that song which was first sung at the cradle," brings pulmonary tuberculosis as observed in the adult back to childhood. With the introduction of Tuberculin as a specific diagnostic measure by v. Pirquet the early infection and subsequent tuberculous disease was again definitely placed in childhood. All this only goes to prove that suitable therapeutic measures directed against the disease must begin in childhood.

Tuberculosis is primarily a child disease, is preventable in childhood, is acquired in childhood, it should be treated in childhood and can be cured in childhood.

In children before the school age much out-of-door life is most essential and during the school age body development must keep pace with mental training. It may perhaps be better not to send

the tuberculous child to school or only for a few hours each day. The instructions at school should be followed by gymnastic exercises and with sufficient rest and sleep, good food, obedience to the rules of health, including hygiene of the mouth, etc.

Primary tuberculous disease, peribronchial gland tuberculosis can with our present knowledge be definitely diagnosed and should receive our closest attention. If peribronchial gland tuberculosis is the first manifestation of tuberculous disease following the implantation of the tubercle bacillus into the human body, and this implantation is in early child life and that all the other forms of tuberculosis are only a sequel of this primary disorder then does it not become our imperative duty to apply therapeutic measures at a period when the disease may become promptly arrested and so guard against a more serious disturbance in later life, at puberty? We must endeavor by appropriate therapy to raise the child's resistance to increase its immunity so that it may safely be carried beyond those years when new impulses contribute to a reactivation of a latent tuberculous process but now attacking a more vulnerable organ, the lungs. Only too frequently this early disturbance is treated very lightly and when the more grave symptoms begin to show, when resistance has been so much lowered then there is no longer an arrest of the tuberculous process possible.

As soon as a diagnosis of bronchial gland tuberculosis has been definitely made, no matter how well the general condition of the child may appear it becomes the imperative duty of the physician to institute a proper therapy. It may be advisable if possible to have the child taken care of at a Preventorium away from the congested districts of a city—out in the open, in the country where under medical care and nurses supervision, and strict obedience to hygienic laws improvement in the child's condition becomes readily noticeable. The child's stay at a Preventorium must be long enough to assure complete arrest of the process after which a periodic examination should be made at least once every three months. This should be done for a period of years or until the child is safely carried beyond puberty. The same rules must apply if the child is taken care of at the home and here the physician must see that strict and general hygiene are instituted similar to institutional care. If all the children, who from the first to fifth or perhaps the sixth year, are victims of persistent and troublesome whooping cough like symptoms, and

who most likely are suffering from peribronchial gland tuberculosis, could be under close observation for many years to follow, we would have fewer cases of pulmonary tuberculosis in young adult or in later life.

(2) Symptomatic Treatment.

Besides living up to strictly hygienic rules much good may be accomplished in the care of the tuberculous child by proper medication. Symptomatic treatment, if necessary, should be applied as the occasion arises, our chief aim in using medicaments must be to increase the child's resistance and to raise its immunity. Children who do not eat well, appear anemic, perhaps cachectic, underfed or undernourished, the addition of some palatable preparation of iron to their daily diet may be advisable. The albuminate of iron and simple syrup in equal proportions make a most admirable combination. This may be given in one-half teaspoonful doses. Less palatable, perhaps more efficient, is the syrup of ferrous iodide and glycerin in equal parts, given in 10 to 30 drop doses three times a day. Although not an iron preparation but one which I have found most satisfactory especially to the very young is the preparation known as Lilly's Coca Quinine. This I direct to be diluted equally with syrup of tolu to be given to a child say about three or four years of age one-half teaspoonful three or four times a day. A remedy which I find most dependable is the ordinary tincture of iodine U. S. P. Five to ten drops to be given in milk three or four times a day, giving it for months, perhaps years. A pleasant stomachic for children is:

Tincture of Nux Vomica drops 16......................(1.0 cc)
Tincture of Cardamom Compound drachms two........(8.0 cc)
Simple Syrup sufficient to make two ounces ....(60.0 cc)

Directions: Give one-half teaspoonful three times a day with meals, in water.

To children with pronounced anemia from two to five drops of Fowler's solution of arsenic may be given three times a day. Cathartics as a rule should not be given. Regulate the bowels by giving the child a suitable diet. A most reliable combination is any of the numerous cereals or breakfast foods now so much in use, with cream and sugar to which a heaping tablespoonful of bran has been added. Give freely of cooked and raw fruit and vegetables. Apples are most ideal if they are

washed and eaten skins and all. Same applies to pears and many other fruit. Milk is the most ideal of all foods. It is best to give it raw and cold, cooking lessens its value, is less palatable, less nutritious. If the milk is from a reliable herd it will be perfectly safe to have the child drink it raw; however, it may be best to pasteurize it. If there should be the least doubt then by all means it must be cooked. Besides milk, good and wholesome bread and butter, crackers, potatoes, meats, ice cream, eggs, etc., should constitute the chief articles of diet for the tuberculous child. As to fats sufficient is secured in the form of butter in the ingested milk or cream, sufficient to supply the necessary calories. Other animal or vegetable fats are unnecessary. Perhaps the addition of a teaspoonful of olive oil several times a day may be added to the daily regime but cod liver oil should be strictly tabooed; it was never intended for a child's stomach.

(3) The Tuberculin Therapy in Gland Tuberculosis—The Specific Therapy.

To tuberculin must be assigned the most conspicuous place for the treatment of peribronchial gland tuberculosis. Ever since its use, it has been observed that its effect, particularly in glandular disease in children may often be called a specific, and palpable cervical glands from bacillary infection by way of the mouth, teeth or tonsils have often been noticed to disappear as if by magic. Children as a rule are very tolerant to tuberculin even if it is given in fairly large doses. The belief entertained by some pediatricians that children do not tolerate tuberculin very well, not as well as adults, is founded upon error, on the contrary they tolerate it very well. The many excellent, even complete curative results which from time to time have been reported following the use of tuberculin in the treatment of glandular tuberculosis, even such as may be accompanied by much swelling and tumefaction, if not actual-breakdown of the gland with open sinus formation may in the main be due to the fact that in the so employed tuberculin a component is found just the suitable antigen for an arrest, inhibition, or perhaps practical cure of the tuberculous process in the gland in a given case, whereas if the same tuberculin is employed in another case it may be followed by only a negative result. The many favorable impressions, even in some cases of complete cure following the use of old tuberculin in glandular tuberculosis, goes to prove that in the con-

tained antigens necessary to bring about this change are constant substances, which are given out during the growth of the bacillus and are present in the protein element of the bacillus and not in the lipoid substance of the organism. This also readily explains why the use of tuberculin in pulmonary cases is not followed by any marked success, this is because these, the pulmonary cases, require an antigen which will inhibit, destroy or dissociate the bacillus, this tuberculin can not accomplish, such a body is not contained in tuberculin. These pulmonary cases are not concerned with any bacillary products but these products are of prime importance in the treatment of glandular cases.

In administering tuberculin hypodermically for curative effect old tuberculin is generally understood and used, beginning with a most minute dose a 1/10 cc of the No. 5 Standard Dilution, this equals one millimilligrams or 1/600,000 of a grain. The hypodermic injection should be repeated once in four days, increasing the dose by 1/10 cc with each subsequent medication. When the individuals "Tonic" dose has been reached the tuberculin may be given once in seven days. This should be continued for some time, then given once in two weeks, then three weeks and ultimately once a month for almost indefinite time, for years. For full consideration of this important topic the reader is referred to Chapter 24 "Tuberculin." Its therapeutic application.

(B) The Treatment and Care of the Metastatic Form of Tuberculosis in Children. The Secondary Form of Tuberculosis.

The treatment of the malignant forms both miliary and meningeal has been given when considering these two types. The treatment of the benign form, bone and joint tuberculosis is usually a surgical one. This treatment is fully considered in Chapter 30—"Tuberculosis of Bones and Joints."

(C) The Treatment and Care of Pulmonary Tuberculosis in Children. The Tertiary Form of Tuberculosis.

This does not differ from that of the adult. As pulmonary tuberculous disease in the child runs a course somewhat similar to that of the adult and as the treatment of adult tuberculosis is fully described in Chapter 19, "The Care of the Tuberculous—General and Medical" and as all that is stated there applies equally to the treatment of this disease in children the physician is referred to that chapter.

As the disease in early child life runs a more acute, perhaps a more rapid course, the child must be under constant observation and the instituted treatment be it dietetic, hygienic, medical, tuberculin or whatsoever must be more intensely and vigorously enforced. It is always advisable to place the child suffering from pulmonary tuberculosis under the care of a competent nurse or still better to be cared for at a Sanatorium where the treatment can be better supervised.

Children suffering from tuberculosis of the genito-urinary tract, of the eye or mucous surfaces, of the peritoneum and the various other tissues and organs of the body should be treated according to the rules given in these respective chapters to which the reader is referred.

The Groups of Glands.

As tuberculosis of the lymph glands and glandular disease in general plays so important a role in child life (89), occasionally in adult life as well, it seems advisable that we study the various groups of glands and their lymph flow in relation to the lungs and the other organs of the body.

We shall first consider the lymph supply to (a) the cervical, (b) to the tracheo-bronchial and then that to (c) the other intrathoracic lymph nodes.

(A) The lymph vessels of the tonsillar region supply the group of glands at the angle of the internal jugular and the anterior facial veins; the lymph flows from there to the middle group of the deep cervical, and terminates not by way of the thoracic duct but as cervical trunks directly into the veins, showing that the deep cervical glands empty into the venous system without any support from the supraclavicular lymph nodes, demonstrating positively that the latter, the supraclavicular glands, do not belong to the regional glands of the pharynx and tonsils. There is no direct communication between the cervical and the supraclavicular glands, however, by means of the numerous anastomosing vessels existing, communication may be established. It has never been indisputably demonstrated that the lymph flow from the cervical glands is towards the region of the apical pleura nor to the tracheo-bronchial lymph nodes.

(B) The tracheo-bronchial glands are the regional and local glands of the pulmonary circuit. They form part of the great regional lymph apparatus of the lungs and all material, either infectious or noninfectious, which finds its way into these glands must come by way of the lungs. These lymph glands, the

tracheo-bronchial, are divisible into two main groups, the pretracheal and the paratracheal. The first group, the pretracheal, also receives lymph from the mucous surface lining the anterior tracheal wall, and the flow may be from there to the laterally situated paratracheal glands, usually, however, to the supraclavicular. The second group, the paratracheal, which is generally designated the tracheo-bronchial, lies in long chains in the furrow between the trachea and the oesophagus. This group is again divided into two subgroups, one of which lies median to the arch of the aorta, the other lateral to the pulmonary artery, that is, between it and the hilum. The lymph vessels from all of this region converge towards the supraclavicular nodes and terminate either in one of the supraclavicular glands or more frequently direct into the vein at the lateral angle of the bulb of the jugularis.

(C) The lymph glands of the lungs are in groups at the angles of the bronchial tree. The lymph flow is towards the hilum and it is gathered in the bronchial glands around and about the bifurcation of the trachea. They are described as the right, left and inferior tracheo-bronchial, the inferior group receiving its supply from the middle and lower portions of both the right and left lung and the lateral groups, the right and left tracheo-bronchial glands, from the middle and upper portions of the lung, from the apices of the lungs the lymph flows from the periphery towards the hilum supplying the nearest lymph nodes. From the tracheo-bronchial glands on either side a few large trunks extend laterally and upwards behind the large veins, to the angle between the subclavian and the jugular, or as may occur on the left side empty either into the veins or into the thoracic duct.

The lymph vessels of the upper portion of the pleura extend back to the intercostal glands, those of the lower and posterior flow towards the glands situated before the spinal column and behind the aorta, emptying into the thoracic duct, and those lymph vessels of the anterior pleura supply the group of glands situated along the internal mammary vein and the artery.

Palpable Glands in Children. It is undoubtedly a sad mistake to diagnose all children in whom we find enlarged cervical glands as suffering from gland tuberculosis. When we speak of gland tuberculosis we generally allude to a disease of the endothoracic glands, to a primary tuberculous gland disease, and not simply to tuberculously infected glands. Cervical adenopathy, a few

enlarged glands in the axilla, or in the inguinal region are very common in children. Every physician knows the great frequency of gland enlargement following chicken pox, as a common occurrence in the exanthemata, common in children suffering from head lice, in eczematous patches about the ears, etc., in all of which the adenopathy may last for years. Cervical gland enlargement may follow infection from the mouth, teeth, tonsils, etc., but this infection is infrequently due to the tubercle bacillus. It is now generally recognized that about 65 per cent of all children at the school age are suffering from cervical gland enlargement, and this is caused from infection from various micro-organisms, but in only about 10 per cent is this infection due to the presence of the Koch's bacillus. Because a child has enlarged glands, chiefly cervical, we have not the right to stigmatize it as tuberculous. Gland tuberculosis is the primary stage of the disease; it follows a secondary infection of the regional glands after the primary infection, but it is not the infection itself. We only too often confuse being infected with being diseased.

It may be of interest to observe at this point that full term infants very exceptionally have palpable axillary glands, and even seven months' children are born without palpable axillary glands. The weight of the child has nothing to do with the presence of enlarged glands, and children nourished at the mother's breast have on an average less palpable glands than those artificially fed. Children suffering from chronic intestinal disturbances have many palpable groups of glands, and every chronic disturbance is accompanied by enlarged glands. In lues and sepsis we usually find a great many palpable glands, but comparatively few in tuberculosis.

The Treatment of Tuberculous Adenitis.

In the treatment of tuberculous adenitis there must be considered: (1) The general constitutional and hygienic conditions; (2) The local treatment to glands and (3) locating and treating the primary point of infection. (193) (194) (195) (196)

(1) **The General Constitutional Condition** of the patient must be determined and any change in mode of living and general hygienic conditions corrected, so as to increase the general body resistance to its maximum. This can only be accomplished by thoroughly examining the entire body and all its physiological functions and institute whatever therapeutic measures necessary to accomplish this end. In other words, the patient should be treated as if an active pulmonary lesion were present in order to secure the quickest and most satisfactory results.

(2) **The Local Treatment.** All cases of tuberculous adenitis, whether cervical or located elsewhere, should receive a prompt and thorough course of radiotherapy, regardless of the stage at which they first come under observation. Over ninety per cent will subside and present a clinical cure under this treatment by converting the lymphoid and granulating tissue into fibrous tissue, occasionally calcification will result. Surgical treatment is never indicated until after a thorough course of roentgenotherapy has been employed, then there will be only a very small percentage that will require surgical interference. In cases where the development is slow, running a painless course, and the glands are separated, a prompt result can be expected within three months, with a fair amount of radiation* once every three weeks.

With the more malignant type of cases where the glands develop rather rapidly and tend to fuse into a large mass, which may show early signs of caseation and abscess formation, very heavy radiation is indicated, which should be limited only by the skin tolerance, in order to get as rapid regression as possible. They yield more slowly than the type first described and will require a longer period of treatment. Exposures are usually made every three weeks over a period of three or four months, and then continued at six week intervals for three or four months more. If caseation and abscess formation develop, it is well to aspirate as much as possible and in this way avoid free drainage, as it saves the patient much time and discomfort. Occasionally the skin will break down, so that free drainage is unavoidable, which will in no way interfere with the radiotherapy.

(3) **The primary point of invasion.** This should be located if possible, and treated. The tonsils and naso-pharynx are frequently found to be sources of the primary lesions. The question of removal of the tonsils is the subject of much discussion at present. A general anaesthetic should be avoided whenever possible, as a slight diminution in the general body resistance may be the direct cause of a rapid extension of invasion to the lungs or the other parts of the body, so when tonsillectomy is decided upon, it should best be done under local anaesthesia. A safer procedure is to treat the tonsillar area and naso-pharynx by X-ray, the object being to retard lymphoid cell proliferation

*It is inadvisable to attempt to describe X-ray dosage, as no uniform standard of measuring the amount of radiation given has been decided upon. The treatment should be put into the hands of a competent radiotherapeutist who will be able to judge the radiation necessary in each case.

and in this way produce a shrinking of the tonsil as well as all other lymphoid tissue with a diminution in size of the crypts and obliteration in some cases.

Too much emphasis cannot be placed on roentgenotherapy of all peripheral tuberculosis, for over ninety per cent of all cases will yield to a properly administered course of treatment, and will save the patient the possible complications which accompany operative interference. The absence of a scar on the neck is also to be desired, especially by female patients. A. R. M.

The following observations by competent pediatrists are most interesting.

The Frequency of Tuberculosis in Children

Death shows the highest point in the first year of infant life. Cornet reports from 10,000 living, who died from tuberculosis as follows:

From 0 to end of 1st year—boys 23, girls 26
From 1 to end of 2nd year—boys 21, girls 21
From 2 to end of 3rd year—boys 12, girls 14
From 3 to end of 5th year—boys 6.9, girls 8.0
From 5 to end of 10th year—boys 4.5, girls 6.0 lowest point
From 10 to end of 15th year—boys 4.9, girls 8.9

Clinical observations are much more frequent in the middle years of child life when the secondary stage of the disease manifests itself in the form of gland, bone and joint disorder which usually do not cause death.

From 0 to 1st year of life 15% show tuberculous lesions at autopsy.
From 1 to 2nd year of life 40% show tuberculous lesions at autopsy.
From 2 to 4th year of life 50% show tuberculous lesions at autopsy.
From 4 to 6th year of life 56% show tuberculous lesions at autopsy.
From 6 to 10th year of life 63% show tuberculous lesions at autopsy.
From 10 to 14th year of life 70% show tuberculous lesions at autopsy.

The tuberculin test applied (after Franz Hamburger) to children considered perfectly healthy is as follows. v. Pirquet test:

From 0 to 1st year the reaction was positive in 0 children.
From 1 to 2nd year the reaction was positive in 9 children.
From 2 to 3rd year the reaction was positive in 20 children.
From 3 to 4th year the reaction was positive in 32 children.
From 4 to 5th year the reaction was positive in 52 children.
From 5 to 6th year the reaction was positive in 51 children.
From 6 to 7th year the reaction was positive in 61 children.
From 7 to 8th year the reaction was positive in 73 children.
From 8 to 9th year the reaction was positive in 71 children.
From 9 to 10th year the reaction was positive in 85 children.
From 10 to 11th year the reaction was positive in 93 children.
From 11 to 12th year the reaction was positive in 95 children.
From 12 to 13th year the reaction was positive in 94 children.
From 13 to 14th year the reaction was positive in 94 children.

or approximately 0% in the first year, 9% in the second year and 94% in the 11th to 14 years.

This was found to apply to the larger cities amongst the poorer population. The wealthier classes show relatively a slightly less infection and this is undoubtedly due to the fact that amongst the poorer classes we find more active or open cases of the disease and the children are found to live in closer contact with the tuberculous adult and in consequence thereof all such children become more readily infected. Hamburger concludes that tuberculosis in childhood is relatively a harmless disease and states that the prognosis becomes progressively better as the age of infection increases. Inactive or latent tuberculosis is most infrequent in infancy or in the first years of child life because nearly all succumb to this early infection and only in very exceptional cases can they overcome the infection. See Chapter 7—"The Course of the Tuberculous Disease." The mortality is always great in early infant life, decreasing as years go by. Mortality 1st year 90% to 100%, 5th year 20% to 30%, 11th to 14th year 1 or 2%. Most individuals are infected in childhood chiefly through the inhalation route, and this leads to the formation of a primary focus in the lung giving a certain degree of relative immunity which lowers or weakens the activity of the bacillary bodies to reinfection.

Mary Hamilton Williams' observation on 3159 school children in 1910 in Worcestershire 16.2% were found affected with pulmonary tuberculosis is worthy of note.

Pulmonary tuberculosis ages 3 to 6—boys, 9.4%; girls, 10.4%.
Pulmonary tuberculosis ages 12 to 14—boys, 11.5%; girls, 18.7%.

She is of the opinion that as age progresses, more children suffer from tuberculous lesions although decreasing numbers die from the results. These findings do not agree with the above given or with our observations. Pulmonary tuberculosis is very infrequent in the school days, or early child life; it only begins to make its presence definitely known as the child approaches puberty. In the early school days other forms of tuberculosis are more frequently seen, pulmonary very seldom.

Some interesting observations from the Stockholm Public Schools 1908-1909.

Out of a total of 25,600 school children, 15,219 between the ages of 8-15 were examined.

Pulmonary tuberculosis, 1.61% (of all children); boys, 1.72%; girls, 1.50%.

Suspicious of being pulmonary tuberculous, 2.21% (of all children); boys, 2.63%; girls, 1.80%.

Pulmonary tuberculosis is less in the younger children between 8-9 years 1.17%.

Pulmonary tuberculosis is greater in the elder children between 14-15 years 2.21%.

Swollen cervical glands in 65% of all children.

Tuberculous glands in only about 10%.

Bone and joint tuberculosis, 0.59%.

About 1/5 of all children were emaciated and about 1/6 of all undernourished. Vol. III, 559.(89)

CHAPTER 24

TUBERCULIN
A PRODUCT OF THE BACILLARY GROWTH AND OF METABOLISM.

Tuberculin is the product of disease producing bacteria, an heterogenous mixture of specific and non-specific substances derived from bacillary growth, bacillary bodies and from the culture media upon which these bacteria are grown. It does not contain living germs, is not a serum like diphtheria antitoxin, nor a lymph like the virus of vaccine. It is distinctly an isopathic remedy, one that may be used in the treatment of a disease by the disease's own specific products. Its action can in no way be increased or modified by similar substances, there are no synergistic remedies known and if its use is at all proposed either diagnostically or therapeutically, a previous contact with this substance has always been anticipated. Tuberculin in itself is not a toxic remedy. It can be administered in large doses without producing any body disturbances. It, however, becomes highly toxic if given to individuals who have had a previous contact with this agent. (51) (52)

From our present knowledge it is evident that not a single so-called tuberculin is of definite chemical composition, nor have we any tangible knowledge as to what the active therapeutic agent in this remedy may be. All the various bacilli of the acid fast group, human, bovine, avian, grass, smegma as well as those found in cold-blooded animals, like fish, turtle, etc., produce during the process of growth and multiplication a tuberculin-like substance, differing only in the amount of its active but still unknown specific body, and on which the value of the various tuberculins depends. They do not differ qualitatively but distinctly quantitatively. All the tuberculins with which we are now familiar, and from whatever source derived, are mixtures of a specific, with more or less non-specific bodies, mostly albuminous substances, fatty bodies, salts, odoriferous principles, etc. Tuberculins from bouillon cultures, from albumin-free media, or more direct, from the bacillary bodies, a bacillary extract, all

are clinically and biologically alike. Up to the present we have not been able either by chemical or biologic methods to isolate or produce the active principle or clinical agent on which this specific effect depends.

What Led to the Discovery of Tuberculin?

After the discovery of the tubercle bacillus by Koch in 1882 he continued his studies of the properties of this newly discovered bacillus as well as of tuberculous material, on experimental animals, arriving after careful and exact observation at certain definite conclusions which under the caption of "Principia" were promulgated as immutable, physiological and biochemical laws. He observed that (1) if healthy guinea pigs were injected, intracutaneously or subcutaneously with a pure culture of a bacillary growth, that at the point of inoculation nothing was noticeable for 10 to 14 days, that, however, after that time a nodule began to form at the point of introduction, which nodule soon showed signs of softening, breaking down and discharging pus and that the nearest lymph glands became enlarged. This so produced ulcer would not heal, but continued discharging pus during the remainder of the little animal's life. (2) If, however, after a period of about six weeks, the same experimental animal was reinoculated with a like amount of the same material as the first, the effect upon the primary sore was quite pronounced, and the secondarily produced ulcer behaved very differently from the first. After the first inoculation nothing was observed for 14 days; however, after the second inoculation, a sloughing ulcer began to form in a few days, healed very readily, and the neighboring gland did not become enlarged.

This second sore, healing quickly, showed a great difference in the tissue reaction in the animal previously inoculated. It also demonstrated that the first inoculation overcame the effect of the second and as the primary infection, now showing a healing tendency, a tendency which was never observed without giving a second inoculation, the second infection definitely showed a distinct effect upon the first. (3) Next, he observed that if dead bacilli were injected into healthy animals, no disturbance was brought about, and at the point of injection only a local suppuration was noticeable, which soon healed, causing no further tissue destruction. (4) If, however, dead bacilli were injected into a guinea pig previously inoculated with live virus the effect

would be quite different; following the injection the animal usually died in from 6 to 48 hours. His further observations were that if only small doses are used, this would cause much destruction of tissue and death, whereas most minute doses caused neither death nor destruction of tissue, and that if infinitesimal doses were given at intervals of two days, much improvement in the primary ulcer followed. This primary ulcer which under ordinary conditions never healed now showed a distinctly healing tendency, the regional or secondarily involved lymph glands became reduced, the animal showed much improvement, and the disease became seemingly arrested. This proved definitely that dead tubercle bacilli, if injected into a healthy guinea pig were non-toxic, that the injection only resulted in the formation of an abscess which soon healed, but that they were highly toxic to previously infected animals, producing death if given in fairly large doses, and apparently curative if given in very minute doses. (5) Owing to the fact that when he injected dead tubercle bacilli into animals, no absorption of the bacilli took place, but that they were expelled with the abscess material, he separated by means of filtration the dead bacilli from the fluid media, and he then noticed that if this so obtained filtrate was used, the results were identical in action with that of the dead bacilli, but without the production of suppuration. This bacillary filtrate since that time, that is, since 1890, when Koch conducted these experiments is known as "tuberculin." This is now prepared as a commercial product, is an artificial tuberculin, and in short is designated "tuberculin" in contradistinction to the product given off by the bacillus while passing through the human or animal body, which is known as the natural tuberculin. It is estimated that the artificially prepared tuberculin, as it is now offered as a remedial agent, contains about one per cent of an active, not yet isolated nor identified protein principle.

The Tuberculin Reaction.

The newly established fact, that this filtrate, the media in which the bacillary bodies grew, proved to be non-toxic if injected into healthy guinea pigs, but highly toxic to inoculated animals if given in larger doses and seemingly curative if given in very minute doses, now received the most intensive study and consideration. After closely observing the effect of this new

remedy on a large number of experimental animals, Koch began experimenting on himself by injecting into his body 0.25 cc of this filtrate, and he became more than surprised with its effect. He observed on himself a rise in temperature, a rapidity of the pulse, a reaction at the point of injection, much malaise, loss of appetite, headache, etc., in short for a time he considered himself made fatally toxic. Nothing like this had ever been observed in man before.

From our present knowledge concerning the almost general tuberculous infection in the human family, we would anticipate such a reaction in the tuberculously infected, but in his time nothing was known concerning the ubiquity of tuberculosis, and he was at a loss either to understand or to explain the reaction, but long before his death he was made acquainted with all the newer problems involved in tuberculosis, and the tuberculin reaction as first observed on himself was then perfectly clear to him.

Explanation of the Tuberculin Reactions.

The so-called tuberculin reaction as we observe it today is usually three-fold: (1) A reaction at the point of application, identified by more or less redness or hyperemia; this is designated the local reaction. (2) The application of the tuberculin may be followed by symptoms of malaise, headache and other constitutional disturbances; this is known as the constitutional, systemic or general reaction, and (3) more or less hyperemia and turgescence about the various tuberculous foci in the body. This is usually described as the focal reaction. That this latter does take place can readily be demonstrated in lupus, where after a tuberculin application a hyperemic area or a reddened line is frequently observed surrounding the lupoid structure.

There are various views entertained concerning these reactions.

(1) **Koch's View or Explanation.** Tubercle bacilli when growing in the tissue produce a substance or substances which kill protoplasm, the death of which brings about tissue necrosis; this necrosis checks the growth of the bacilli in consequence of which many die. If now tuberculin is administered, it will favor the production of large necrotic areas, and this causes many more bacilli to die, and if now these tuberculin injections are frequently repeated, more and more bacilli will be destroyed until ultimately all are killed, when the process becomes healed and the reaction ceases.

(2) **The Wolff-Eisner Theory** (28). When tubercle bacilli are deposited in the tissues of the human body and begin to show activity, that is, growth and multiplication, antagonistic forces are inaugurated. These forces are of two kinds, one of which is to destroy the bacilli, the other to neutralize the toxic products given off in this destruction. The first kind or class of antagonistic substances are known as amboceptors or lysins, bacteriolysins, the functions of which are to break down and digest the bacilli, converting them into simpler as well as into complex and toxic bodies; the function of the second class of substances is to neutralize these complex poisonous bodies, and these in general, are spoken of as antibodies. Lysins or amboceptors not only destroy tubercle bacilli, but tuberculin, the toxic product of bacillary growth, as well. Tuberculin is non-toxic to healthy individuals, even if given in large doses, but it is highly toxic to most tuberculously infected and many diseased individuals, and in such persons the injected or applied tuberculin is seized by the lysins or amboceptors present in the organism, the result of which is the conversion of the injected tuberculin into a tuberculo-lysin, which is now highly toxic. This lysinized tuberculin causes a reaction at the point of application, at the site of injection, circulating through the system producing a general or systemic reaction, and acting as a toxic irritant on the tuberculous centers, a focal reaction. If this lysinized tuberculin is now completely neutralized by the second substance, the antibodies, then no reaction follows.

(3) **Ponndorf's Law** (100). Ponndorf, sanitary physician at the Variola Institute at Weimar, reports most interesting experimentations. His observations relating to immunity in smallpox vaccination established certain notable facts, namely, that as the skin and mucous membrane of every person offer most suitable protection to the body against mechanical insults of all kinds, examples of which are daily observed by the external influences of heat and cold, so they offer also a protection to the organism after infection from various microörganisms and their products; further that the elaborated specific toxins, the result of bacterial infection and subsequent growth and development are taken up by the epithelial layers of both the skin and mucous membrane, and there with more or less rapidity reduced, changed or dissociated into simpler and less toxic substances, and that a subsequent infection or contact with the same microörganisms or their

toxins will produce, with the chemically changed contents or modified toxins of these cells, new combinations, which manifest themselves in the production of an inflammatory reaction at the site of application and by a generalized systemic disturbance. He then made analogous observations on the skin and mucous membrane of tuberculous individuals. After infection with the tubercle bacillus there will be found, at all times, deposited in the epithelial cells of the skin and mucous membranes modified, changed or dissociated natural tuberculin, and the great biochemical affinity existing between this modified tuberculin in these cells and the now added artificial tuberculin brings about a chemical reaction. This view seems most plausible. It is well known that the bacilli and toxin free organism does not react to tuberculin, this because when artificial tuberculin is applied to the skin of an individual who has never been infected, it does not find modified or changed, systematically produced tuberculin with which to enter into chemical union, hence no reaction. On the other hand, the toxically tuberculous individual also does not react to this applied tuberculin because the tuberculin present in the epithelial cells of the skin in such persons is unchanged, not modified, and being identical with the artificially produced product, no biochemical change can take place within these cells.

The Various Kinds of Tuberculins.

Up to the present time there are offered to the medical profession in round numbers about 100 different tuberculins. This in itself speaks for the great uncertainty and diversified composition of this preparation. The therapeutic value and cutaneous reaction of all depend upon a supposed biochemical toxic substance present in each in varying amounts. The following in the order given are the best known tuberculins now in use:

(1) **Old tuberculin O. T.** Koch's old tuberculin, the original tuberculin of 1890, also known as extract tuberculin. Preparation: A pure live virulent culture of the tubercle bacillus of human origin in a 5% glycerine nutrient broth is incubated for 5 or 6 weeks, after which it is sterilized for one-half hour, evaporated at 70° C to one-tenth its volume, filtered and 0.5 of pure phenol added. Tuberculin so prepared contains from 40 to 50% glycerine, 10% peptone or albuminose, toxic substances, the derivative of the bacillary growth, and bacillary extract derived from the process of heating and evaporation.

(2) **Deny's boullion filtrate. B. F.** This preparation is very similar to the former, the old tuberculin, differing only in that in preparing it no heat is employed, hence it contains less bacillary extract.

(3) **Tuberculin. B. O. T.** Nathan Raw and Carl Spengler suggested a tuberculin after the manner of the O. T. preparation, inoculating the culture media, the broth, with a virulent strain of the bovine bacillus. It is in every other particular identical with the original old tuberculin.

(4) **New tuberculin. T. R.**=tuberculin residue, 1897. Endoplasm turberculin. Koch's reasoning led him to the conclusion that in the use of old tuberculin the object is the obtaining of an immunity against a tuberculin toxicity and not a true immunity against the bacterium, hence, his aim was to produce a product which would give to the organism when applied a bacterial as against a toxic immunity. With this object in mind he offered in 1897 a new tuberculin now generally known as T. R. This is prepared as follows: Dried, virulent tubercle bacilli of the human kind are thoroughly triturated and ground with distilled water then centrifugalized and the supernatent watery fluid rejected. The residue remaining in the centrifuging tube is dried, again triturated, ground with distilled water and centrifuged, the process being continued until all of the bacillary mass has been taken up by the water. 20% of glycerin is then added to the impalpably powdered bacilli held in suspension by the distilled water. This preparation is of such standard strength that each cc=2 milligrams of solid substance or 0.002.

(5) **Bacillen emulsion. B. E.** A combined tuberculin, both endoplasm and bacillary extract, 1901. After four years' use of new tuberculin, a second preparation of tuberculin. known as second new tuberculin was advanced by Koch. This is now known as bacillary emulsion or simply B. E., a combination of endotoxin and exotoxin. It is supposed to bring about when understandingly applied both a bacillary and a toxic immunity. It is prepared by suspending 0.5 gram of the impalpably powdered tubercle bacilli in 100 cc of equal parts of glycerine and distilled water. This is then repeatedly shaken until an emulsion is effected. Each cc=5 milligrams or 0.005. It is not a bacillary emulsion but simply a bacillary suspension of bacilli in an impalpable powder in a suitable vehicle.

(6) **Polyvalent tuberculin.** A polyvalent T. B. emulsion. Poly-

valent autotuberculin. A combination of 8 different strains of various virulence, like an autogenous vaccine. It fell quickly into disuse and at the present time is but little employed.

(7) **Tuberculin Bèranéck, T. B. K.,** an extract and endoplasm tuberculin. This tuberculin is a mixture of a culture of the human kind, free from all tubercle bacilli, evaporated at low temperature in vacuum to a syrupy consistency, and a bacillary extract obtained by treating bacillary bodies with a 1% solution of orthophosphoric acid, without evaporation. This tuberculin presents a bacillary protein in the form of a phosphoric acid albumin combination. The culture media used is free from peptone and albuminous substances, furnishing a tuberculin free from non-specific proteins.

(8) **Spengler's I. K.** (Immune Koerper.) Immune bodies. The value of this preparation is based upon the supposed facts demonstrated by Carl Spengler, namely, that in the red blood corpuscles, the erythrocytes, of every infected human being, precipitins, tuberculo-precipitins are present which in a dilution of 1-10,000 can be recognized. Specific tuberculin treatment will increase these bodies (1-1,000,000 and more). The amount of these precipitin bodies in the human organism is variable; they are naturally increased with the increase of resistance to infection.

(9) **Albumose-free tuberculin T. A. F.,** a tuberculin produced or prepared by cultivating tubercle bacilli on a media free from foreign albumin. The non-specific albumin content of the ordinary or old tuberculin and to which action many of the undesirable reactions have been attributed is thereby wholly eliminated.

(10) **Endotoxin. Tuberculinum Purum.** Gabrilowitch. Offered under the name of Tuberculinum Purum, this tuberculin is supposed to be free from all foreign proteins. It is prepared by the use of chemical reagents like alcohol, ether, chloroform, xylol and by precipitation, washing, decantation, centrifugalization, etc., from albumose—free tuberculin.

(11) **Partialantigens or Partigens.** Much, Deyche, Leschke. These co-workers have demonstrated that the tubercle bacillus by means of diluted organic acids, lactic, malic, phosphuric, etc., can be dissociated or separated into its component parts and that these separated parts consist of a free fatty acid, a lipoid substance, a toxic principle, a neutral fat, a protein product, various salts, etc. Their extensive research has proven that the antibodies

produced in the tuberculous individual are not of a simple nature but that different defense agencies are produced, differing from those following the injection of pure tuberculin, from those following the incorporation of the neutral fat, the fatty acid or the other toxic components of the bacillus. They have demonstrated that by means of the complement fixation test and by testing the hypersensitiveness with these separate components, it is possible in every case of tuberculosis to show which partial-antigens are present and which are not present and it is only necessary to ascertain in each case which are present in or absent from the organism and then to supply such as are needed to effect a favorable result, that each component part when injected into an animal or the human body is capable of producing distinct, definite and specific antibodies in the organism so injected, that these bodies or antigens are specific for each individual antibody and that the tuberculously infected person with a pulmonary lesion is in dire need of these antigens to produce in the infected organism the necessary antibodies to cope with the invasion. If all are absent, the combined partialantigens must be employed to bring about the necessary antibodies.[1]

(12) **Friedmann's (89) Prophylactic and Curative Vaccine,** (so-called) or the Friedmann Remedy. This dates back to 1902 and 1903 when he experimented with a bacillus passed through a turtle (161). These organisms are supposedly avirulent; live, natural bodies. If they are human bacilli attentuated by repeated passage through cold blooded animals, like a turtle, or if they are really cold blooded animal bacilli, has not been determined satisfactorily. Nothing definite is known, it is still in the experimental stage and while it finds some friends it has also found many who are not friendly, however, from the literature, we have learned that what can be achieved with this remedy can be se-

[1]The tubercle bacillus is dissociated under the influence of lactic acid into a soluble (L) and into an insoluble (R) Antigen. The reaction of the soluble (L) is dependent upon the toxin hypersensitiveness and the insoluble or (R) upon immune body sensitization. According to Much, the soluble portion (L) of the bacillus is very fatal to guinea pigs, not so the insoluble (R) or residual portion. The soluble (L) portion is a pure tuberculin. The insoluble (R) portion is further devisible into products designated respectively A. F. & N. With R or the products of R, the body's immunization activities can be increased and the toxin hypersensitiveness decreased. A specific odoriferous body has also been isolated.

cured with the products of the ordinary tubercle bacillus. In 1913 the Friedmann Remedy was first heralded as a tuberculosis cure since which time and up to the present it has been extensively used by many clinicians of repute, still we have no proof of its special curative properties.

(13) **The Serum Therapy.** (1) **Maragliano's Serum.** An attempt at a cure or arrest of the tuberculous process by means of passive immunization, by serum therapy. Maragliano maintains that it is made possible by means of serum injections to produce specific antibodies and also to immunize the organism against tuberculosis. Horses or cattle are treated first with avirulent and then later with moderately virulent tubercle bacilli. After a prolonged treatment a serum is secured similar to the method in use for preparing diphtheria antitoxin.

(2) Marmoreck's Serum. A serum similar to the above but with slight modification, which has had a fairly good run of success. It has been proven that in a measure it is possible by passive immunization to inhibit the tuberculous process.

The use of Tuberculin—(a) therapeutically; (b) diagnostically. Indications and Contra-indications. (72) (75) (76) (79).

(A) Therapeutic Indications

In the use of tuberculin for therapeutic purposes, the object desired is to bring about a destruction, if possible, of the tubercle bacillus, to inhibit its growth, to stop the toxin formation,—in short to bring about bacteriolysis.

Tuberculin, like all other therapeutic agents, has its limitations and can be used in some cases for good, in others, and this by far the greater member, for bad; hence, it more often has no place in the treatment of tuberculosis. If the human organism is supplied with more than sufficient tuberculin from within, that is, produced within the body, then it is absolutely useless and even harmful to supply more from without; this means that each case must be carefully studied before we give tuberculin. The therapeutic use of tuberculin is usually indicated in two classes of cases:

1. **In the Presence of Localized Colonies.** Here the lesion is generally not active, and there is little or no extension of the process, hence little or no production of tuberculin within the body. As a consequence there is no absorption of toxin, no toxin in the circulating blood and no body changes. This we find

usually in tuberculosis of the glands, in bone and joint disease and in lupus. With such lesions the process generally does not heal because body changes have not taken place, no bacteriolysis being present, or if present, insufficient in amount to check the slow extension of the process. In such cases the use of tuberculin is indicated to excite the production of lysins.

2. **In Generalized Tuberculosis.** If autotuberculin absorption is taking place, tuberculin is contraindicated and must not be used because there is already too much tuberculin from within in the body. This is usually in cases having night sweats, high temperature and rapid pulse, where with rest and quiet a lessening of tuberculin absorption may be brought about to control an autotuberculin absorption.

In beginning cases, old tuberculin may be used successfully if not accompanied by much fever. Bacillen Emulsion is generally indicated in second and third stage cases; hence, tuberculin may be used in the beginning or in progressive cases if we always carefully bear in mind what we desire to accomplish.

When, How and How Long Should Tuberculin Be Administered?

Different views are entertained by various therapeutists as to the length of time that a tuberculin medication is to be continued.[2] There exists also a difference of opinion as to the reaction; should tuberculin be given with (52) the aim of producing a slight reaction or should we always stay within the limits, strictly avoiding a reaction.

Petruschky begins with small minute doses, gradually increasing until fairly large doses are given. This he continues for a period of three months, after which for a like period no tuberculin is administered. Then, in a similar manner, tuberculin in fairly large doses is again given for three months and discontinued for three months and so on for a period of two years.

Sahli begins with minute doses, the initial dose being 1/100 mg., gradually increasing the dose till large doses are given, avoiding at all times a local reaction and a rise in temperature; however, should there be a rise in temperature during the treatment, the dose must be lessened by one-half the dose which produced the disturbance. The treatment may then be continued in

[2] A person who is hypersensitive to tuberculin if treated with fairly large doses may have such a severe reaction as to result in most dire consequences. An active and hypersensitive tuberculous individual if given a 1/20 millimilligram or less of tuberculin, may show within six to twenty-four hours a distinct systematic disturbance, malaise, rapidity of the heart's action, depression of spirits, rapid respiration with increased expectoration, in fact a sudden and severe aggravation of all symptoms.

this manner for a period of months and even years or the treatment may be discontinued for some time and then again resumed.

Lowenstein, among others, entertains opposite views. He advocates the giving of tuberculin to a point of producing a reaction. We do not yet know if the giving of tuberculin which is followed by a reaction is favorable to connective tissue formation, as is so vigorously maintained by some, or if it favors the propagation and extension of the tuberculous process, as is the opinion of others; however, it is a much safer and wiser plan to stay within the limits of a reaction, to avoid reactions; large doses are dangerous whereas small doses are often very beneficial.

There may be a pseudo lessening of the temperature curve following the use of fairly large doses of tuberculin, but a pulse frequency, loss in weight, dyspnoea and nausea give evidence of the liberation of large amounts of endotoxins from bacteriolysis, all indicative of overstimulation.

As a therapeutic remedy for the treatment of tuberculosis, tuberculin can not be considered a specific. It is no more a specific remedy than are the other agencies in use. The dietetic-hygienic, the fresh air and all other remedies at our command are just as specific as any in aiming to bring about active immunization.

In the treatment of tuberculous disease, when the process is still localized, the method as first suggested by Wright finds its best application. Here the object is to produce a mild immunization without tolerance. By this, the Wright method, tuberculin is first given in minute doses every 4th or 6th day, the dose being gradually increased until a dosage is reached at which the individual expresses a feeling of well being, after which this same dose is maintained for some time. This is the patient's proper dose, "the tonic dose." In generalized tuberculosis, however, the method of Koch is the one usually employed. This is giving tuberculin with tolerance; by this method tuberculin is administered progressively until enormous amounts are given even up to the point of giving pure tuberculin. At the present time most therapeutists are giving tuberculin in all and every form of tuberculous disease according to the former, the Wright method, selecting the "Tonic dose" which may be given for long periods, almost indefinitely, for months and even years. In administering tuberculin we must bear in mind that the ob-

ject sought is the bringing about of a toxic immunity, an immunity against the patient's produced body tuberculin but not against his tuberculosis, because we do not make him tuberculosis immune.

If, in cases of generalized tuberculosis, tuberculin is used at all, it should be given in the interim between the exacerbations and remissions. If at all it is then most effective and if beneficial, is followed by an immunizing response, a feeling of well being. The infected individual as well as the actively diseased is generally very sensitive to tuberculin, is tuberculin sensitized; this must always be borne in mind when using this agent.

In the use of tuberculin for therapeutic purposes all forms have been tried from old tuberculin down to the newer sera. When given as a curative remedy, tuberculin as an initial dose is usually applied in the most minute dilution. The introduction of tuberculin into medicine was hailed as a remedy of great promise; it was thought that at last a remedy had been discovered which would cure tuberculosis. The fiasco following its induction as a therapeutic agency is still fresh in the memory of many, but gradually after a better understanding concerning the action and possibilities of this valuable drug, it found its proper level, and at the present time it is chiefly used in therapy, as an additional measure for the control of this disease, mainly as a help as a tonic or stimulant in the treatment.

Tuberculin in itself never has, never can and never will cure a case of tuberculosis, but it is a most valuable adjunct in the fight, if aligned with fresh air, good food and out-of-door life, etc. In specially selected cases its use is undoubtedly indicated. The beginning dose must always be a small one and the dosage must be most accurate. The method of administering is with the hypodermic needle. Whereas tuberculin for diagnostic purposes is given cutaneously, percutaneously and intracutaneously, for therapy it should be given subcutaneously, under, and not into the skin.[3]

The Methods of Administration

After having selected a case of pulmonary tuberculosis for treatment, or for that matter any form of the disorder, we usually

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[3]Occasionally to active tuberculous individuals tuberculin is given subcutaneously in fairly large doses to test the patient's tolerance, his sensitiveness. This should not be construed as giving tuberculin diagnostically. In such instances the diagnosis has been definitely made, no need of tuberculin to aid us in the diagnosis. Here the test is only given with the object of testing the patient's tolerance to tuberculin, his immunizing activities.

begin by giving a very small quantity, say 1/20, 1/10, 1/2 or perhaps in exceptional cases 1 millimilligram as a first or initial injection. The treatment is repeated at first every 4th day, giving 2/10, then 3/10 then 4/10 millimilligrams and so on, always with gradually increasing dosage. After 14 or 16 injections have been given, the remedy should be administered every 7 days, and after continuing the treatment for some time the interval between injections may be lengthened, giving a treatment once in 2 or perhaps 3 weeks, or the treatment may be entirely suspended for a while, say for a month or two when a similar progressive course may be given. If during the treatment, say after a dozen or more doses have been administered, a reaction should follow the last injection, then it is usually customary to drop back to half the dosage which caused the reaction and give that amount at the next treatment, then gradually increasing the dosage until fairly large doses are given, watching the effect after each treatment. Always remembering that the use of tuberculin for therapeutic purposes is directly in opposition to the activity of the disorder, that if the disease is pursuing a very slow, a more or less protracted course with little or no fever, pulse not very rapid and showing a tendency to fibroid tissue change, the therapeutic use of tuberculin may be of great value; if, however, the tuberculous process is very active or exacerbations and remissions follow each other in rapid succession or if the disease shows a very stormy tendency, the tuberculin must not be used—is strictly contraindicated.

Tuberculin in the treatment of pulmonary tuberculosis has not always fulfilled all the anticipated requirements of a therapeutic remedy. We must remember that the tuberculously infected and the diseased individual suffering from an active, chronic disorder is very frequently not in need of any more antigens because his own body supplies him with an amount sufficient over and above all requirements of his body in the fight. Here the body is not in need of more corpuscles, possessing protein splitting properties, but it requires above all such agencies as will produce lipolysis, and the stimulus or antigens necessary for that is not contained in the tuberculin, either old or new; hence, the great failure of a tuberculin impression being followed by very favorable results.

We recognize usually three phases of tuberculin administration: (1) the tolerant; (2) the stimulating and (3) the toxic or exciting. In mild cases, a slightly positive tuberculin reaction is a favorable sign. If the reaction during the process of the disease becomes negative, this also is a favorable indication; how-

ever, in progressive cases a negative reaction presages an approach of death. A tuberculin reaction indicates that the reacting individual is either already clinically tuberculous, that is, diseased, or that a previous infection has immunized him, and although a reaction is present he is clinically healthy.

Occasionally while using tuberculin therapeutically, even in very minute doses, the injection may be followed by a very severe reaction, and we must not at once conclude that the subject is hypersensitive. In the use of tuberculin as is now in vogue, six separate dilutions of this powerful drug are by common usage recommended, each one ten times stronger than the one next in order, and it is more than evident that in the use of such a potent remedy if we should give to a suspected person one milligram tuberculin equal to 1/60 grain for diagnostic purposes and then a moment after, using the same hypodermic syringe, give to another patient one-tenth of a millimilligram equal to 1/600,000 of a grain that untoward effects may manifest themselves; hence, a separate syringe should be used for each separate dilution.

Begin the Treatment with Very Minute Doses

The question has often been asked why we begin with such minute, infinitesimal doses in order to safely influence the tuberculous person. I cannot do better than to direct your attention to a graphic picture which I have so often used to explain this phenomenon:

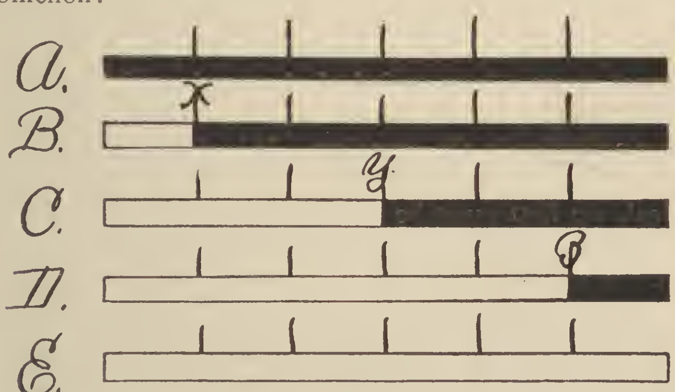

Fig. 43. Illustrating the degree of tuberculin tolerance between four infected individuals when compared with the non-infected.

Let the lines, A, B, C, D and E represent five patients, and let A represent a person who has never been infected with the virus, while B, C, D and E represent tuberculously infected individuals. A, never having been infected is tolerant to enormously large doses, is not sensitized and will tolerate from one to ten centimeters or more of any of the solutions in our series, but not so B, C, D or E, who have been infected, and having been infected, they are more or less sensitized, but we do not know up to which point or to what dosage. We know A's tolerance, but we have up to the present time no positive knowledge about the tuberculin tolerance of either B, C, D or E, the infected persons. Let us assume then that B's tolerance ceases at X, and if by any sure means we could ascertain that, we could begin our tuberculin medication a little short of that point and gradually encroach on sensitiveness, but we have no absolute way of knowing this in advance, and for that reason we must begin at the extreme end and give the most minute quantity, and slowly and gradually advance the dose. C's tolerance is at Y, and D's at Z, and the same applies as in B's case. E is also tuberculously infected, but is so much sensitized that he will not tolerate the most minute quantity of our dilution of 1-1,000,000, and we can encroach on his sensitized state only by repeated minute injections and that at long intervals; it is for this reason that in all the four given cases of infected persons the initial therapeutic dose should be infinitesimally small so as to ascertain the individual's tolerance; when once that has been established correspondingly larger doses may be given.[4]

Tuberculin is a most valuable and reliable therapeutic agent for the treatment of tuberculosis if properly administered. Its use must cover a long period of observation. It is not a cure, does not bring about true immunization, but does produce stimulating activities in the organism. It is simply a most valuable adjunct in our armamentarium. In giving tuberculin to an individual, we first must be sure that he or she is really tuberculous. The statement has been made by some therapeutists that those patients usually do best, are more readily healed, under tuberculin medication in whom the tubercle bacillus can not be demonstrated, that is in the closed and not in the open cases.

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[4]During the course of tuberculin therapy, the cutaneous reaction often becomes negative. This then is an indication that the tubercu'ous process is becoming arrested. With a reactivation of the process, however, the skin reaction may again become positive, hence during specific treatment the dermal reaction may become alternately positive and negative or vice versa.

but here the question arises whether they actually had tuberculosis, and even with physical signs present it may still leave a doubt.

Various Other Therapeutic Methods for Administering Tuberculin

The giving of Tuberculin therapeutically by means of the hypodermic syringe (subcutaneously) is not the only method of tuberculin medication. There are many other serviceable methods now in vogue, some of which may be equal or even superior to a subcutaneous injection. The Mantoux or intradermal method, so successfully used in the application of tuberculin for diagnostic purposes, may also be applied to the principle of treatment. Produce a Mantoux upon the cleansed cutaneous surface of the upper arm of the tuberculous individual using $1/10$ cc No. 5 Standard Solution and carefully note the result of this first application. If a slightly hyperemic area is noticeable then in seven days from the time this first intracutaneous injection was given, a similar amount or $1/10$ cc No. 5 should again be given. Should the second cutaneous medication prove similar to the first then in seven days give another $1/10$ cc, No. 5. This dosage of $1/10$ cc No. 5 may be repeated a number of times and be continued until no inflammatory area is noticeable at the point of application when with the next medication we use 0.1 cc, No. 4 and proceed as with No. 5. By this method the tuberculous individual often becomes rapidly tolerant to fairly large doses of tuberculin, expressing a feeling of well being, not so quickly acquired by the ordinary subcutaneous injection method. This treatment may be maintained for some time and after an interval or period of rest a second similar course may be given. It is not necessary to continue with the treatment until large doses are given. I have never given a dose higher than $1/10$ cc, No. 3. The same rule applies here as in all other methods of tuberculin medication, that is, if during the course of treatment the intracutaneous injection is followed by much inflammation, perhaps also systematic disturbances then it is advisable to discontinue the method altogether for some time or drop back to a lower dilution. This method of tuberculin medication has proven most satisfactorily in treating tuberculous disease of the eye, skin, cervical glands, joints, etc.

Within current years the use of tuberculin for therapeutic purposes by the Ponndorf Method of Cutaneous Vaccination (83) has found much favor, and judging from the available literature, the results appear most promising, the method most rational, even to a point of supplementing all the other methods of treatment. The upper arm of the tuberculous individual is thoroughly cleansed, sponged with alcohol, then scarified with an ordinary v. Pirquet scarifier or preferably a hypodermic needle with not too sharp a cutting edge. Make upon this so prepared surface of the skin from 10 to 20 or more linear scarifications about 2 (5 cm) inches in length (parallel lines) and about $1/16$ (2 mm) inch apart. Do not scarify deep nor draw blood, just sufficient to denude the surface of the skin and upon this so prepared area place a drop of O. T. or B. C., or T. R., or any form of pure tuberculin and with the middle finger of the right hand, well protected with a sterile rubber finger cot, rub the tuber-

culin well into the abraded surface. A glass rod with a rounded end or a small test tube will answer the purpose just as well. Now cover this tuberculinized surface with waxed paper securely held in place by a few strips of adhesive plaster allowing it to remain for 24 hours. A word of precaution. It is well known that very sensitive individuals will react to most minute quantities of tuberculin, hence a cutaneous vaccination using pure tuberculin may be followed by most distressing consequences. In order to avoid such unpleasant occurrences, it is best first to test the individual's tuberculin susceptibility; this can readily be accomplished by making a v. Pirquet or a Mantoux before beginning the tuberculin therapy by the vaccination method. If the probationary test is very positive, then the cutaneous medication must be given very cautiously, probably making only a few scarifications and using only a minute quantity of tuberculin, or better, postpone the treatment for a month however, if the probationary test is found fairly positive then in about one week after, the vaccination may be made. These vaccinations should be repeated but once in 21 days or even at longer intervals and usually not more than 10 such scarifications are necessary. The results obtained by this method of treatment are often more than phenomenal.

Another method of tuberculin medication should here be mentioned. One first suggested by Petruschky, namely a 1% tuberculin, glycerin solution. A drop of this mixture is placed upon the previously cleansed skin of the arm, back, or chest, is well rubbed in and no covering applied. A drop may be so applied every other day for an indefinite period. I have seen most satisfactory results follow this form of medication when applied to small children suffering from tuberculous peritonitis, in tuberculous skin lesions, in tuberculosis of the cervical gland and for the after treatment in surgical tuberculosis.

(B) Contraindications in the Use of Tuberculin

Much harm can be done if tuberculin is used in cases in which it is strictly contraindicated. Tuberculin should never be used if in a tuberculous individual the fever is very high, showing toxin absorption, nor if the tuberculous process is massive, showing great pulmonary involvement, or in suspected mixed infection. It should not be used if both lungs are the seat of much disease, if the disease is pursuing a rapid course, the pulse very rapid, nor in hemorrhagic cases. In diabetes and nephritis, if complicating pulmonary tuberculosis, the use of tuberculin is always contraindicated. In tuberculous subjects suffering from an accompanying neurasthenia, epilepsy or cirrhosis of the liver, tuberculin must not be used. It is very dangerous to use tuberculin if measles, influenza or pneumonia preceded the active tuberculous process. In diffuse tuberculosis without expectoration, tuberculin as a therapeutic measure must not be employed, and tuberculosis and cardiac complications are always signs for

inhibition; hence, in all the above enumerated conditions it is advisable not to use tuberculin; it is strictly contraindicated.

Tuberculin for Diagnostic Purposes

Although tuberculin as a therapeutic agent for the treatment of tuberculosis has its sharply defined limitations, in its use as a diagnostic or probationary remedy it finds its widest application. The characteristic value of tuberculin as a specific remedy depends upon the reaction produced on the cutaneous or mucous surfaces of the tuberculously infected. As previously stated (see page 311, this chapter) this reaction results from the presence of modified natural tuberculin in the epithelial cells of the skin, which, having great affinity for the added tuberculin, by biochemical changes produces a hyperemic area at the point of application; hence, in order to bring about a reaction, modified, natural (or artificial) tuberculin must be in the individual on whom the test is applied; the person must have come in contact with the virus, or its toxins at some previous time.

In a person who has at no time come in contact with the tubercle bacillus or its products no reaction[5] will follow even if very large doses of tuberculin are given, showing that it has toxic properties only in the infected organism and that it is perfectly non-toxic in the non-infected. It is in this respect that it possesses specific actions and it is in this sense understood when we refer to tuberculin as a specific remedy.

Various kinds and different quantities of tuberculin are used for diagnostic purpose, some using tuberculin in its purity, others using it in the different dilutions.

The Various Tuberculin Tests

Four approved tests are now in general use.

1. **The Ophthalmic or conjunctival test.** The application of tuberculin as a diagnostic measure to mucous surfaces is usually in the diluted form, and the mucous membranes of the nose, ear and vagina have all been used, but that of the eye alone has been found convenient. This is now generally known as the Calmette test. By means of this method, also known as the

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[5]In order to produce a tuberculin reaction in the organism, the foci of infection must be so situated that the products of metabolism of the invading bacilli have entered the circulation, producing what is now generally known as a positive reaction. This condition is in pulmonary tuberculosis almost always fulfilled, but not always so in the other tuberculous disorders. If there is no destruction nor products of bacillary disintegration and growth to enter the circulation, then there is no tuberculin reaction. The small amount of tuberculin applied favors encapsulation of the glands, arrest of the necrotic process, healing, and under strictly hygienic and dietetic treatment, improved and an increase in body weight.

Wolff-Eisner, a drop of diluted tuberculin 1-100 is instilled onto the conjunctiva of one eye, the other being used as a control. In a few days the treated eye will be quite hyperemic, swollen and irritated, while the untreated eye remains normal. This is known as the Calmette reaction. Clinically a positive conjunctival reaction points with great probability to an active tuberculous process, however, as a sure diagnostic sign it must not be considered. This method is now somewhat in disfavor and is but little employed; in fact the mucous surfaces are very little used when compared with applications of tuberculin to the cutaneous surface.

The application of tuberculin to the derma for diagnostic purposes is known as (1) the Moro, the percutaneous test; (2) the v. Pirquet, the cutaneous test; and (3) the Mantoux, the intracutaneous test.

2. **The Moro or the percutaneous test.** A 50% ointment prepared by mixing intimately equal parts of lanolin and tuberculin O. T., is applied to a previously prepared surface of the skin. An area about 5 cm in diameter over the chest, abdomen or upper arm is thoroughly cleansed with soap and water, then with alcohol, and when dry the Moro ointment about the size of a split pea is rubbed well into this prepared surface and covered with waxed paper for 24 hours. A reaction, said to be positive, is followed within 24 to 48 hours by a reddened area slightly elevated, throughout which many or few more or less raised papules are noticeable. The reaction usually lasts a few days, gradually fading away and leaving a slightly pigmented spot. For me the Moro test is the one of choice in all small children up to about the fourth year.

The tuberculin reaction in infants and small children is of great clinical importance. A positive reaction in a child does not tell us if the case is active, but in a child up to the second year the reaction generally indicates an active tuberculous process, because in children at that age the disease is always active.

3. **The v. Pirquet or the cutaneous test.** This test is usually applied by making three slight scarifications on the upper or forearm, the cutaneous surface having been first thoroughly cleansed. A specially devised scarifier is generally used, but any instrument with not too sharp a cutting edge may be substituted. The scarifications should be made just sufficient to produce a slightly hyperemic point, just sufficient to denude the skin, just below

the horny layer. Three scarifications are made in a row usually from 2.5 to 3 cm apart and to two of these, generally the outer, the first and third, a minute quantity of pure tuberculin is applied, the central scarified point being used as a control. This is now covered with waxed paper for 24 hours. In a positive v. Pirquet, an hyperemic zone 1 cm or more is usually noticeable at the points of application, the control showing little or no inflammation. As occasionally may happen when the scarifications are made too deep, the tuberculin applied areas as well as the control may show hyperemic areas of about equal size, which is due simply to the irritation to the skin, and must not be construed as a positive test. In such an event, a second test must be made at an interval of about a week.

A positive v. Pirquet usually shows a hyperemic area about the size of a dime, the control being negligible. The v. Pirquet test is applicable in children from the 4th year up to the 12th, and perhaps to puberty but not beyond. The v. Pirquet in young children may be positive in only about 45% of cases to a first application and 65% to a second, while the next, the Mantoux, is positive in nearly 80% to a first test.

4. **The Mantoux, the intracutaneous or intradermal test.** Is also described as the Esherich or Römer. This test is generally made by injecting into and not under the skin a definite quantity of tuberculin in salt solution. The amount for an initial dose is generally a milligram, 0.001. Many diagnosticians give as an initial dose, 1/10th of a milligram and if no reaction is noticeable in 3 days, 1/2 milligram; if again there is no reaction, a full milligram, continuing as high as 10 milligrams; if still there is no reaction, the individual is pronounced non-tuberculous. Most adults react to a dose of 1 milligram, 0.001, a second injection is usually not necessary, should, however, no reaction follow this dosage, then the use of 2 or more milligrams is justifiable. If the tuberculin has been well deposited into the epithelial cells of the skin, generally in the tuberculously infected individual, a reaction will be recognizable which is distinct and specific. Usually within 12 to 48 hours, occasionally as late as 72, at the point of application a reddened area from 1.5 to 3 or more cm will be observed, and the area is generally surrounded by a distinct halo of lessened hyperemia in size from 5 to 8 or more cm. To the palpating finger the perception of a distinct induration under the skin often referred to as a "button" is imparted. The reaction

usually begins about six hours after the injection reaching its greatest intensity after the second day. The induration may last a few days or evidence may still be noticeable in a few weeks. Usually, with a very positive Mantoux there is great hyperemia at the point of application, the temperature rises and the pulse is quickened somewhat, the patient complaining of malaise, want of appetite, perhaps a slight headache, all of which are recognized as more or less systemic disturbances, the accompanying cough giving evidence of the focal reaction. It goes without saying that the skin should be cleansed before giving an intracutaneous injection—alcohol usually sufficing. A typical Mantoux should be given, as has been so aptly stated by Carl Spengler, in a centrifugal manner, the skin being slightly raised and pitted, giving it the appearance of being blanched; and the injected tuberculin should lie about the point of the needle so that the point lies in the center of the produced wheal. The giving of tuberculin by mouth for diagnostic purposes should here be incidentally mentioned. Give old tuberculin in pill form in 0.05 gram dose if not systemic reaction is noticeable, then, after three days, give 2 or 3 pills of the same strength, and if no reaction follows this dosage, an intradermal injection should be given. By this method the previously prepared organism responds much more readily to the tuberculin if given intracutaneously.

The Negative Tuberculin Reaction

The tuberculin reaction is negative in three classes of cases, first, in individuals who never came in contact with the virus and consequently have no defense agencies or antibodies in their blood plasma, second, in those who at some time in their lives have come in contact with the virus, and either not sufficient time has elapsed since the infection for the body to produce antibodies, or for some reason the body is not putting up a defense, perhaps possesses a definite immunity and is wholly indifferent to the invaders. Again, the reaction is negative in cases of active and advanced disease, much cachaxia, in such, the defense powers have long been exhausted, the test applied too late, long after the body was capable of enlisting the defending forces against the invaders. In such individuals the artificial tuberculin used for the test and the natural tuberculin in the body are alike, hence no reaction. In the first two instances the test was applied too early and in the last too late to be of diag-

nostic importance. It is only when the body shows that it is disturbed by the invaders, is trying to throw off the infection, when the infection has aroused the body's defense powers in either a limited or in an extensive degree, that the test becomes positive. Of greatest importance is the negative reaction, because this may eliminate tuberculous disease, with the exceptions already mentioned above.

To this may be added a fourth. It is a well known fact that in the exanthemata the tuberculin test is generally negative. A child just beginning to show prodormal symptoms of measles, or even before, if tuberculin is given intracutaneously a negative reaction will follow, this, then, may be utilized as a positive measle test.

In this connection it may become necessary to call attention to the following questions often referred to:

What is meant by a distinctly positive tuberculin reaction? The tuberculin test is positive[6] whenever the defense agencies or antibodies are freely present in the economy (a good prognosis). These antibodies are most abundant in the first years of infection, after an attack of recurring tuberculosis and after a reinfection, either from without or from within. A positive reaction does not state what stage is present, whether a beginning stage or a remission. If clinical symptoms point to a tuberculous process, a positive tuberculin reaction, simply confirms this suspicion. In infants the tuberculous process is less latent; hence, here a positive reaction usually indicates a tuberculous disease.

What does a mildly positive reaction indicate? It indicates that there has been a previous infection but that the antibody content of the organism is not very extensive (a less favorable prognosis). This is frequently found in healed processes; it may

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[6]The Nature of Resistance to Tuberculosis (164)

(1) Reaction occurs only when anatomic tubercle is present in the body;
(2) The reaction begins to manifest itself with the establishment of the tubercle;
(3) Up to a certain point, as the tuberculous involvement or disease progresses, the intensity of the reaction increases;
(4) As the tuberculous disease heals or retrogresses, the intensity of the reaction lessens;
(5) As tubercles probably are never completely eradicated from the body, the power to react is probably never completely lost. Hnce the following corollary:
(1) The presence of the tubercle endows the body with power to greatly resist increased numbers of newly added bacilli.
(2) This increased resistance to reinfection manifests itself with the establishment of the primary focus.
(3) Up to a certain point, resistance is proportionate to the extent and severity of the initial disease.
(4) With the healing of the diseased foci, resistance diminishes.
(5) If tuberculous disease remains, the increased power to resist is probably never lost entirely and does not drop to the level before first infection. (Allen K. Krause.)

also indicate an old progressive condition (tuberculous). Tuberculin in adults frequently gives but a mild reaction. The reaction following a previous subcutaneous tuberculin injection (secondary reaction) is of this order. No reaction, no sensitiveness in miliary tuberculosis or in measles, croupous pneumonia as concomitant disorders, in tuberculous meningitis nor in cases incorporated with large doses of tuberculin. Most tuberculous individuals to whom tuberculin is administered in the usual way never react to the dosage; some only react when fairly large doses have been given.

In giving tuberculin diagnostically and that applies to its use for therapy as well, the patient must be under the constant observation of the physician. Tuberculin to be used intelligently must be diluted with the greatest accuracy and I will next describe in detail this method of dilution.

Tuberculin Dilutions

The necessary apparatus for preparing the proper dilutions of tuberculin are few; one-half dozen 10 cc bottles, a graduated 10 cc cylinder, a pipette or medicine dropper, a quantity of a normal salt solution to which ½ of 1% of phenol has been added and a small amount, say 1 cc of pure tuberculin.

In using the word tuberculin, O. T. or old tuberculin, the original tuberculin is generally understood, but any of the other kind may be used. After the vials which are intended to receive the different dilutions have been thoroughly cleansed, rinsed with distilled water and dried, we will be ready to proceed, granting that the pipette, the graduated cylinder and the other necessary apparatus are also perfectly clean. Place into the 10 cc graduated cylinder 1 cc of tuberculin and add to it 9 cc of the phenolized salt solution and after all is well mixed pour it into one of the clean bottles at hand and label it No. 0. This is a 10% tuberculin dilution, it is a stock solution and is seldom used for either diagnostic or therapeutic purposes. Next pour 1 cc of No. 0 into the 10 cc graduated cylinder and dilute it with 9 cc of phenolized salt solution, place it into a clean bottle and label it No. 1. This is a 1% solution, the dilution most frequently used for diagnostic purposes. Now take 1 cc of No. 1, dilute with 9 cc of salt solution and label it No. 2; this is a 1/10 of 1% solution. 1 cc of No. 2 and 9 cc of phenolized salt solution makes No. 3, a 1/100 of 1% solution. Again 1 cc of

No. 3 and 9 cc of salt solution makes a 1/1000 of 1%, or No. 4, and 1 cc No. 4 with 9 cc of salt solution is known as No. 5, equaling a 1/10,000 of 1% solution. No. 5 usually furnishes the initial doses when tuberculin is given therapeutically. A 1/10 cc of this solution No. 5 equals 1/100,000 of 1%, being the first dose. Occasionally it is advisable to begin with still smaller doses, when 1/20 of a cc is given as the beginning dose. In labeling the bottles from 0 to 5, no special reason can be given for this designation; a 1 to 6 labeling may be just as proper, it is only that originally I began making dilutions from 0 to 5 and this I have ever since maintained.

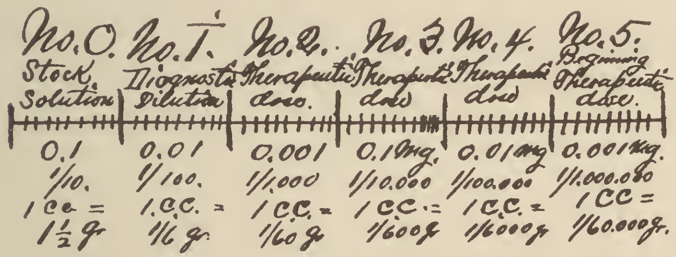

Fig. 44. Tuberculin Dilutions. Standard Scale.

As to dosage, begin by giving 1/10th cc of No. 5 and observe whether there is any reaction, always remembering that extremely hypersensitive individuals may react to a most minute dose; if there is no reaction, in about 4 days, give 2/10 of No. 5, always watching; after 4 days more give 3/10, 4 days more 4/10, and so on until 9/10 cc of No. 5 have been given. Now with the 10th treatment we do not give a full cc of No. 5, but 1/10 cc of the next dilution, No. 4; this is because 1/10 of No. 4 equals a full cc of No. 5; by giving the former we simply lessen the amount of fluid to be injected. We now continue in a similar manner with the solution No. 4 as we did with No. 5, perhaps giving the injection less often, say once in 5 or 7 days until the 9th dose of No. 4 has been given, when with the next dose we give 1/10 cc of No. 3 in place of a full cc of No. 4. We continue with No. 3 in like manner and by this progressively increasing dosage it is often possible to reach a dosage of pure tuberculin in full cc of No. 0. This, however, is not advisable. If the patient is tolerant to a dosage when No. 2 is reached, it is good practice to discontinue all further treatment for a few months and then repeat the medication.

In giving tuberculin therapeutically, we aim to be within the limits of a reaction, within tolerance, for diagnostic purposes, however, our purpose is to bring about a reaction. If during the course of tuberculin therapy the patient is doing well, the pulse rate somewhat reduced, temperature within the normal for a tuberculous individual, and he expresses a feeling of well-being, then the dosage at the time this is observed may be maintained, giving this same dose once or twice a month, or perhaps only monthly. **This then is the individual's tonic dose.**

It must become apparent to anyone administering tuberculin that after giving the drug for a comparatively short time after this method, enormously large doses may be given. As the amounts increase in 10s in each dilution and as each is 10 times stronger than the next lower (higher number), when No. 2 is reached, we are giving 1000 times the dosage, when compared with the initial dose. In using tuberculin dilutions according to the plan advocated by Prof. Bèraneck, the dilutions rise in potencies of 2 instead of 10; hence, the treatment may be carried on over much longer periods and more gradually. The accompanying table is self-explanatory:

Bèraneck Scale

	Decreasing Scale		Increasing Scale	
H	= T.B.K.	(Bèraneck Tuberculin)	A/16384	Bèraneck T. Dilution
G	= T.B.K./2	Bèraneck T. Dilution	A/8192	Bèraneck T. Dilution
F	= T.B.K./4	Bèraneck T. Dilution	A/4096	Bèraneck T. Dilution
E	= T.B.K./8	Bèraneck T. Dilution	A/2048	Bèraneck T. Dilution
D	= T.B.K./16	Bèraneck T. Dilution	A/1024	Bèraneck T. Dilution
C	= T.B.K./32	Bèraneck T. Dilution	A/512	Bèraneck T. Dilution
B	= T.B.K./64	Bèraneck T. Dilution	A/256	Bèraneck T. Dilution
A/1	= T.B.K./128	Bèraneck T. Dilution	A/128	Bèraneck T. Dilution
A/2	= T.B.K./256	Bèraneck T. Dilution	A/64	Bèraneck T. Dilution
A/4	= T.B.K./512	Bèraneck T. Dilution	A/32	Bèraneck T. Dilution
A/8	= T.B.K./1024	Bèraneck T. Dilution	A/16	Bèraneck T. Dilution
A/16	= T.B.K./2048	Bèraneck T. Dilution	A/8	Bèraneck T. Dilution
A/32	= T.B.K./4096	Bèraneck T. Dilution	A/4	Bèraneck T. Dilution
A/64	= T.B.K./8192	Bèraneck T. Dilution	A/2	Bèraneck T. Dilution
A/128	= T.B.K./16384	Bèraneck T. Dilution	A	Bèraneck T. Dilution
A/256	= T.B.K./32768	Bèraneck T. Dilution	B	Bèraneck T. Dilution
A/512	= T.B.K./65536	Bèraneck T. Dilution	C	Bèraneck T. Dilution
A/1024	= T.B.K./131072	Bèraneck T. Dilution	D	Bèraneck T. Dilution
A/2048	= T.B.K./262144	Bèraneck T. Dilution	E	Bèraneck T. Dilution
A/4096	= T.B.K./524288	Bèraneck T. Dilution	F	Bèraneck T. Dilution
etc. if desired			G	Bèraneck T. Dilution
			H	(Tuberculin Bèraneck)

A ½ syringeful of any dilution equals in amount a whole syringeful of the next, in the decreasing scale. In using Bèraneck's tuberculin, each cc is again divided into 1/5 cc (or 1/10 if desired), giving 5 doses before giving the next higher solution.

The approximate value of Bèraneck's tuberculin B. T. K. and the various dilutions is expressed in cc, grams, grains and fractions thereof, as follows:

H	= B.T.K.	1 cc	= 1 grm.	= 15	grs.	(approximately)
G	= B.T.K./2	1/2 cc	= 1/2 grm.	= 8	grs.	(approximately)
F	= B.T.K./4	1/4 cc	= 1/4 grm.	= 4	grs.	(approximately)
E	= B.T.K./8	1/8 cc	= 1/8 grm.	= 2	grs.	(approximately)
D	= B.T.K./16	1/16 cc	= 1/16 grm.	= 1	gr.	(approximately)
C	= B.T.K./32	1/32 cc	= 1/32 grm.	= 1/2	gr.	(approximately)
B	= B.T.K./64	1/64 cc	= 1/64 grm.	= 1/4	gr.	(approximately)
A	= B.T.K./128	1/128 cc	= 1/128 grm.	= 1/8	gr.	(approximately)
A/2	= B.T.K./256	1/256 cc	= 1/256 grm.	= 1/16	gr.	(approximately)
A/4	= B.T.K./512	1/512 cc	= 1/512 grm.	= 1/32	gr.	(approximately)
A/8	= B.T.K./1024	1/1024 cc	= 1/1024 grm.	= 1/64	gr.	(approximately)
A/16	= B.T.K./2048	1/2048 cc	= 1/2048 grm.	= 1/128	gr.	(approximately)
A/32	= B.T.K./4096	1/4096 cc	= 1/4096 grm.	= 1/256	gr.	(approximately)
A/64	= B.T.K./8192	1/8192 cc	= 1/8192 grm.	= 1/512	gr.	(approximately)
A/128	= B.T.K./16384	1/16384 cc	= 1/16384 grm.	= 1/1024	gr.	(approximately)
A/256	= B.T.K./32768	1/32768 cc	= 1/32768 grm.	= 1/2048	gr.	(approximately)
A/512	= B.T.K./65536	1/65536 cc	= 1/65536 grm.	= 1/4096	gr.	(approximately)
A/1024	= B.T.K./131072	1/131072 cc	= 1/131072 grm.	= 1/8192	gr.	(approximately)
A/2048	= B.T.K./262144	1/262144 cc	= 1/262144 grm.	= 1/16384	gr.	(approximately)
A/4096	= B.T.K./524288	1/524288 cc	= 1/524288 grm.	= 1/32768	gr.	(approximately)

Ad infinitum.

The Standard Tuberculin Dosage

In administering tuberculin therapeutically the standard dilutions mentioned should be given in gradually increasing dosage. This increase must be a steady one so as to avoid any untoward disturbances such as are occasionally observed in the treatment. Tuberculin, being a remedy of great potency, may produce very alarming symptoms in an individual who is not tolerant to the drug. In using the ordinary or standard dilutions now in general use, only too frequently very large, even enormous amounts are given after comparatively few injections. If in a given case we begin the treatment, as is usual, with a very minute dose, say 1/10 cc of No. 5, which is equal to 1/10th of a millimilligram, and then with each succeeding dose we gradually increase this amount, we will, when given the 19th injection, have administered a dosage equal to 100 times that of the initial dose; hence, the intervals of dosage must now be lengthened and the results carefully observed. Note the rapid increase:

The 1st injection of No. 5 or	1/10 cc	= 0.000.000.1	= 1/10 mmg.
The 2nd injection of No. 5 or	2/10 cc	= 0.000.000.2	= 2/10 mmg.
The 3rd injection of No. 5 or	3/10 cc	= 0.000.000.3	= 3/10 mmg.
The 4th injection of No. 5 or	4/10 cc	= 0.000.000.4	= 4/10 mmg.
The 5th injection of No. 5 or	5/10 cc	= 0.000.000.5	= 5/10 mmg.
The 6th injection of No. 5 or	6/10 cc	= 0.000.000.6	= 6/10 mmg.
The 7th injection of No. 5 or	7/10 cc	= 0.000.000.7	= 7/10 mmg.
The 8th injection of No. 5 or	8/10 cc	= 0.000.000.8	= 8/10 mmg.
The 9th injection of No. 5 or	9/10 cc	= 0.000.000.9	= 9/10 mmg.
The 10th injection of No. 5 or	10/10 cc	= 0.000.001.0	= 1 mmg. but

one cc of No. 5=1/10 cc of No. 4, and so instead of giving a full cc of No. 5 we give only a 1/10 of a cc of No. 4 as its equivalent and then we proceed as before.

The 10th injection is from No. 4 (1/10 cc)=0.000.001; this equals in amount 10 times the quantity given with the first injection.

The 11th injection is 2/10 cc from No. 4 = 0.000.002. This, however, does not equal 11 times the initial dosage but 20 times that amount.

Next, the 12th injection from No. 4 = 3/10 cc = 0.000.003 = 30 times and not 12 times the initial dosage, and

the 13th injection from No. 4 = 4/10 cc = 0.000.004 = 40 times (the initial dose)
the 14th injection from No. 4 = 5/10 cc = 0.000.005 = 50 times (the initial dose)
the 15th injection from No. 4 = 6/10 cc = 0.000.006 = 60 times (the initial dose)
the 16th injection from No. 4 = 7/10 cc = 0.000.007 = 70 times (the initial dose)
the 17th injection from No. 4 = 8/10 cc = 0.000.008 = 80 times (the initial dose)
the 18th injection from No. 4 = 9/10 cc = 0.000.009 = 90 times (the initial dose)
the 19th injection from No. 4 = 10/10 cc = 0.000.010 = 100 times (the initial dose)

but 10/10 cc (or 1 cc) from No. 4=1/10 cc from No. 3, and if we proceed as before, then the 28th injection would be 10/10 of No. 3 or 1/10 of No. 2. This amount would equal 1000 times the first or initial injection. The 37th[6] injection would be 10/10 of No. 2 or 1/10 of No. 1, and this amount would equal 10,000 times the first dose. The 46th injection would equal 100,000 times the initial dose and 1/10 cc the 55th injection 1,000,000 times the initial dose or pure tuberculin.

For table of Standard Tuberculin Dilutions, see Page 542.

[6]Note: The students' attentions are specifically directed to these figures. They are intended to illustrate here simply the rapid rise in the tuberculin dosage and not intended for actual use or medication. Such enormous doses never have and never can be given; this would be a dangerous and an irrational and impossible therapy.

CHAPTER 25

TUBERCULOSIS AND PLEURISY

Tuberculous Pleurisy. Pleuritis Tuberculosa. Idiopathic Pleurisy. Pleuritis Idiopathica

General Consideration. (47) If a tuberculous pleurisy manifests itself without accompanying inflammatory conditions then only the formation of tubercules is present. This is often observed in generalized miliary disease as an accompanying miliary tuberculosis of the pleura. An idiopathic tuberculous pleurisy in the course of chronic pulmonary disorder is the most frequent phenomenon. In this two forms are recognized—a dry or sicca and a moist or humida. (a) The dry form favors the apices, the paravertebral areas and at the bases the lateral regions (borders). This may be either circumscribed or cover quite extensive areas. In this the exudate is generally fibrinous and may be reabsorbed or as most frequently happens, connective tissue scar formation takes place, producing adhesions of the two pleural surfaces, or if much thickening results, may undergo calcification or extensive adhesions between which more or less fluid may be sacculated. (b) In the other, the moist, the fluid may be either serous, sero-fibrinous, purulent or hemorrhagic. The serous fluid in the pleural cavity is usually the result of an inflammatory process of toxic origin and not due to the presence of the tubercle bacillus. In the sero-fibrinous form fibrinous flakes, yellowish in color, are mixed with the serum, and in this fluid tubercle bacilli are usually demonstrable. The quantity of serous or sero-fibrinous fluid may vary from one hundred to many thousand cc; in fact, the fluid in the pleural cavity may be sufficient in amount to compress the lung completely, dislocate the heart, liver spleen and diaphragm, more or less adhesions between the lungs and other viscera take place and if the fluid is incompletely absorbed, new tissue develops consisting of connective tissue and blood vessels, the inevitable consequence resulting in a pleural thickening of 1 to 2 cm. A purulent tuberculous pleurisy, a tuberculous empyema, results either from the

presence of the tubercle bacillus or from pus germs in the pleural cavity, however, it frequently happens that the pus is sterile, that is, free from all foreign organisms. In such instances the presence of the pus is usually the result of a chemical toxin.

The Frequency of Tuberculous Pleurisy. A great diversity of opinion exists among the various authors as to the frequency of a tuberculous pleurisy. Many good observers maintain that perhaps only about 10% of all pleurisies are tuberculous, while, on the other hand, equally good clinicians state that nearly all are, and Landouzy among others opines that at least 98% are tuberculous, whereas Osler has made the terse expression, "that the more I see of pleurisy the more I become convinced that they are all tuberculous." However, that may be, close observation has undoubtedly proven that idiopathic pleurisies, in more than 95% as a rule, are followed by active tuberculous disease within the first five years. Tuberculous pleurisy it may be stated, is always or perhaps most frequently secondary to a pulmonary tuberculous disease. It must be admitted that pleurisy may accompany tuberculosis of the bronchial glands, Pott's disease, a tuberculous peritonitis, may even be hematogenous, etc., and a possibility of a primary tuberculous pleurisy may be anticipated from penetrating thoracic wounds, carrying infection directly into the pleural cavity. In all those cases in which an apparent idiopathic pleurisy existed previously to a pulmonary tuberculosis, the lung disease was either latent or it was not earlier recognized. Men are more prone to a tuberculous pleurisy than are women in the proportion of 2 to 1, and seasons also play a very important role, there being more in the midwinter months and less in the fall.

Symptoms. In miliary tuberculosis of the pleura the clinical symptoms are negative so long as a plastic inflammation or a serous exudate is absent. An apical pleurisy is usually a pleuritis sicca. A dry pleurisy shows the characteristic symptoms of localized pain on pressure and a dry cough and the pain is increased on breathing in consequence of which the excursion of the chest is limited. The patient endeavors to suppress the cough and at the same time makes inward pressure over the involved pleura, or he lies on the afflicted side. In simple idiopathic pleurisy with little pulmonary activity, fever is usually absent or else very slight, and a dry pleurisy if accompanied by much pain for some time is usually quickly followed by the

exudative or moist form; hence the course of the process may vary, may remain of the dry, or be followed by the moist variety, or the pleura may remain thickened without any adhesions, or both surfaces of the pleura become adherent. The most frequent seat of a pleuritic inflammation, the seat of predilection is in the axilla.

The course of the disease. This varies greatly. In the dry form it may remain stationary for years, or retrograde, leaving only a slightly thickened pleura, or the visceral and parietal pleura may become adherent, or it may eventually assume the exudative form. The exudative form usually begins with a pain or stitch in the side, which is very much aggravated by coughing, sneezing, and breathing and as the amount of fluid increases in the pleural cavity, separating the two surfaces of the inflamed pleura, the pain subsides (indux and redux) but the dyspnea now generally increases, the severity of the shortness of breath depending upon the rapidity with which the fluid accumulates and upon the quantity of fluid in the pleural cavity. If, on the other hand, the fluid accumulates slowly dyspnea may be entirely absent even with large amounts of exudate. Pleurisy may not be suspected until a physical examination reveals the true nature of the disorder—the patient complaining only of a tired feeling, loss of appetite, and some gastro-intestinal distress. This usually in the subacute form of the disease.

Physical Examination

Inspection. On inspection we observe a lessened or impaired motion on the affected side, and owing to the pain on respiration the patient usually lies on this side to suppress as much as possible the movements of the chest. The intercostal spaces suffer very little change and although it is frequently stated that with a large exudate in the pleural cavity the intercostal spaces bulge, I have never observed it.

Palpation. On palpation we observe an absence of vocal fremitus and a sense of fulness or inelasticity is imparted to the palpating finger; the intercostal muscles feel spastic.

Percussion. The note is flat immediately over the fluid, dull above and becoming more or less resonant and tympanitic as we proceed upward, depending upon the amount of fluid in the pleural cavity. The percussion sounds change but little with the changed position of the patient, this is because the fluid is

not free to move about in the chest cavity but is held in position by the inflamed and adherent pleural surfaces about the fluid; however, if the patient remains for some time in the changed position the fluid will be found to have changed also. The fluid in moist pleuritis is usually at a higher level in the axilla and lower toward the spine as well as toward the sternum. This was early pointed out by Damoiseau, Ellis, Garland, and others.

Auscultation. In pleurisy it is most distinct and definite. In recent pleurisies and while the pleura is still thin, the sound conduction is very clear but this changes gradually as the pleura becomes thickened from fibrinous deposit, and from the increase of the exudate, and while vesicular sounds are not audible over the pleural fluid, bronchial sounds become most distinct and the whispering voice is that of whispering bronochophomy. In this respect it may simulate pneumonia very much, from which, however, it can readily be differentiated by the absence of the tactile fremitus. The compressed lung above the fluid also gives bronchial breathing, increase in the vocal fremitus whilst towards the apices increased vesicular sounds may be heard, and often a high and tympanitic note may be elicited on percussion and over the upper portions of the exudate and where the fluid layers are thin the egophonic voice may be heard. If the exudate is profuse the heart may be found dislocated or crowded toward the healthy side, the diaphragm is usually lowered and in right sided pleurisies the liver as well.

Diagnosis. An early diagnosis is of the greatest importance in pleurisy although in only too many instances the first symptom which alarms the patient is the difficulty in breathing for which he consults the physician when the chest is already found greatly filled with fluid. Pleurisy, both the dry and the moist form, is usually of tuberculous origin, secondary to a tuberculous process in some part of the organism. It is a well known fact that microorganisms other than the tubercle bacillus may cause a pleuritis, however, this is infrequent. A latent tuberculous process in a lung may show activity many years after a pleurisy, it is for this reason the imperative duty of the physician who is taking care of a case of pleuritis to inform the patient that this disorder may sooner or later be followed by active pulmonary tuberculosis, the same applies to pneumonia and to the so-called walking typhoid cases as well. The subjective symptoms usually observed are pain in the chest, stitch in the lung, sideache,

pressure symptoms and neuralgia-like apical pain. Tuberculous patients who are free from pain generally have an intact pleura. In the dry form of pleuritis a friction murmur simulating the creaking of leather is audible and frequently palpable as well. These pleural friction sounds are in general audible during the whole inspiratory phase and sometimes during the expiratory and subcrepitant râles are audible at the apices, usually at the end of the inspiratory act, if the bronchi are free and the air currents can reach the bronchiols and alveoli. Friction sounds are nearer to the ear more superficial than murmurs produced in the lungs proper.

Differential Diagnosis. Fine crackling sounds heard over the lungs at their borders or in the axillary lines from the 5th to the 8th rib may be either of pleural origin or are due to atelectatic or emphysematous changes in the lung proper. These latter are differentiated from pleural sounds which are heard through the whole of the inspiratory and part of the expiratory phase, by being heard with the height of inspiration only, they are produced in the pulmonary parenchyma. Harsh, interrupted or systolic breathing may be confused with dry pleurisy, but the pulsating or cardiac rhythm gives the differentiation. With pleural adhesions the friction sounds cease. Apical pleuritis is often very difficult to recognize. Friction sounds are usually absent and an early obliteration of the costal pleura the rule. Muscle sounds may simulate pleurisy and pain may be entirely absent or very slight, not especially noticeable by the patient when changes on the chest are observed, such as a slight prominence of the clavicle or the suprascapular fossa, or posterior muscle spasm or slight shoulder pain, perhaps a definite pulmonary involvement showing that an apical pleurisy may be suspected. In pleurisy with effusion the diagnosis as a rule is much easier, although cases are not infrequently encountered in which the diagnosis offers many difficulties. In moist pleurisy the fluid may appear either very slowly or the pleural cavity be filled with a fibrinous exudate in a very short time. It may be accompanied by a sharp lancinating stitch in the side as is usually observed in the acute form or it may be wholly unknown to the patient, as in the subacute form, where often the pleural cavity is found nearly filled with an exudate and the one symptom of shortness of breath on exertion leads to an examination.

A paravertebral dulness, Grocco's sign (1902) also called

Rauchfuss' sign (1904) demonstrable along the spine on (89) the healthy side if the pleural cavity of the opposite side contains much fluid. This dulness is present if the exudate reaches the height of the 8th dorsal vertebra. With lessening of the fluid the dulness disappears. This dulness is in the form of a triangle with the apex along the spine indicating the height of the fluid, the broad end of this triangle corresponding to the upper surface of the diaphragm. The cause of this triangular dulness accompanying an exudative pleurisy is the change in the percussion note along the spine from the normal. The fluid is present only on one side of the spine—none on the other. In percussing the thoracic vertebrae from above down, that is, from the first to the tenth, the note normally is usually loud and somewhat resonant but osteal above and below these points. This is due to the adjacent air containing lung structure on both sides of the spine. If fluid is on one side in the pleural cavity the note changes and as the exudate compresses the lungs, the note becomes gradually more flat as the fluid increases and we reach a lower level, the dulness extending more and more to the opposite or healthy side—the dulness along the fluid entirely overshadowing that of the normal side. That this paravertebral dulness is not due to any mediastinal displacement has been corroborated by numerous roentgen examinations. If rapid absorption of the exudate takes place, Grocco's sign is absent. In croupous pneumonia, although the lung is consolidated, the signs of paravertebral dulness are absent on the opposite side perhaps due to the conduction which is good in pneumonia and poor in pleuritis.

The differentiation between lobar pneumonia and pleurisy. In both the note is dull or flat on percussion but in the former the vocal fremitus is increased while it is diminished or absent in the latter, moreover in pneumonia the onset is sudden, usually with a chill and high fever, soon followed by rusty sputa; in both there is pain, but the most distinct single diagnostic point is the whispering voice which is heard over both the consolidated area and over the pleural exudate but over the latter only while the pleura is still free from fibrinous deposit; bronchial breathing is also heard over both but somewhat more intensely and nearer in pneumonia than in pleurisy, but there is a great difference as to fremitus which is very pronounced in pneumonia and absent in pleurisy. If the fluid has persisted for

some time and the surfaces of the pleurae become thickened, both the whispering voice and bronchial breathing disappear and an entire absence of all sounds is noticeable. Baccelli's sign that the whispering voice is distinctly transmitted through serous fluid and not through purulent is not dependable. This also depends upon whether the pleura is thin or thickened from fibrinous exudate.

Exploratory Puncture and Paracentesis. The time has long since passed when a positive diagnosis of pleurisy with effusion was made immediately after the fluid was withdrawn. It is perhaps permissible, nay advisable in doubtful cases, to make an exploratory puncture and withdraw a few cc of fluid to confirm the diagnosis. The question has often been asked when is it advisable to withdraw the fluid from the pleural cavity? The general rule is that if the patient is in great distress, is extremely dyspneic and the fluid fills the greater portion of the pleural cavity, sufficient fluid should be withdrawn to make the patient comfortable. If, however, the patient is quite comfortable and is neither dyspneic nor distressed, it is best to leave matter to nature. We notice that frequently after a certain amount of fluid has accumulated the effusion becomes limited, after which reabsorption often begins. Again, in other instances, although the accumulated fluid is great the patient is not at all distressed, absorption does not take place at once and perhaps only after a long period does a gradual lessening of the fluid become noticeable. It has been observed that only large pleural exudates exert a beneficial influence on the tuberculous process in the lung and that a small amount or very little fluid is detrimental. A tuberculous pleuritis in the course of pulmonary tuberculous disease often exerts a very beneficial influence on the diseased process both in advanced and in beginning cases, all of which goes to show that we must consider well when to puncture or whether to withdraw fluid at all.

Where to Puncture. Best in the 7th intercostal space posterior axillary line, in the 8th or 9th intercostal space midscapular line or in the midaxillary line in the 6th interspace. Here the average diameter of the adult chest wall, at these points is about 4 cm. These punctures should always be made close to the upper border of the rib so as to avoid the important intercostal vessels and nerves.

Prognosis. The miliary form is very grave. The dry form

gives a more favorable prognosis. Here usually adhesions between the two surfaces of the pleura take place. In the sero-fibrinous form the prognosis is equally good. Pleuritis, in every case of pulmonary tuberculosis, if fairly large, exerts a favorable influence on the tuberculous process if the pleurisy is on the same side[1] as is the pulmonary disorder. By compression of the lung structure and pulmonary rest, it presents a tendency to heal the tuberculosis; in fact, the favorable influence exerted on the tuberculous process by the fluid in the thoracic cavity as early observed by the profession led to the artificial induction of lung compression by means of nitrogen gas or filtered air and it is for this reason that the fluid if not troublesome, if not accompanied by much fever, no less in weight nor profuse perspiration or complications, should remain until gradually absorbed. The fluid in the pleural cavity may for some time remain serous or more often sero-fibrinous or it may assume gradually a muco-purulent character, and when conditions become much more grave, an empyema may develop which if evidenced by constitutional disturbances indicates operative interference.

Medical Treatment of Pleurisy. Pleuritis, as stated, may be ushered in abruptly, very acutely, or it may not be known to the patient until shortness of breath compels consulting a physician, when the nature of the disorder is recognized. In this latter form usually little medication is indicated but in the acute form remedies for the relief of symptoms are sought early. The most distressing symptom is the pain or stitch in the side. The patient should rest and sleep lying on the affected side—absolute rest. He must be made comfortable, the distress and nervousness relieved, and for this purpose the use of opium in any form or one of its many derivatives is indicated and morphia sulphate is usually resorted to, being given hypodermically in 1/8 to 1/4 grain doses. In cases in which the symptoms are less acute, the use of local remedies applied directly to the chest over the seat of pain are very efficient, and here the use of mustard is most desirable, preparing a slight mustard draught or a plaster consisting of one tablespoonful of ground or powdered mustard mixed with four or five tablespoonfuls of flour made into a paste with some warm water and of sufficient consistency to allow it to

[1]Many internists maintain that in the majority of instances the tuberculous process usually manifests itself on the same side as the pleural involvement, that is, on the side of the pleurisy. If in latent tuberculous disease where it often follows the pleurisy in years after, then usually the active process in the lung is found on the opposite side of the old pleuritis. However, if the pulmonary process is active at the time when pleurisy is a concomitant disturbance then it is generally on the same side as the pulmonary disorder.

be spread evenly upon a clean piece of cloth to cover a surface of about 6 by 6 inches or larger if desired. A plaster so prepared should remain firmly applied to the chest for about twenty minutes after it begins to smart, then removed, the skin dried and covered with a layer of olive oil and a bandage applied. The use of a tablespoonful of pure and finely ground mustard well mixed in a quart of hot water, a towel wrung out of this warm solution and at once applied to the affected side often gives prompt relief. A topical remedy of great value is the use of ordinary tincture of iodine U. S. P., painted over a large area on the affected side; allow it to dry and cover lightly with a cloth but do not apply a bandage. Both the use of mustard or the tincture of iodine or any other counterirritant must not be used too frequently, say not oftener than once in two or three days, because if used too freely the skin over the affected area will become blistered, adding more to the discomfort. If the skin is normal or has not been irritated too much by the application of local remedies, the use of adhesive straps is a most desirable procedure, these to be applied in strips of about two inches in width extending from the middle of the sternum to the middle of the spine and from a point above the fluid down to and below the costal arch and ribs. With the use of adhesive plaster we aim to immobilize the ribs and prevent the intercostal muscle from contracting during the respiratory act. Such adhesive strips must be applied after the patient expels as much air as possible and then holds the breath. To promote absorption of the exudate many remedies have been recommended, particularly such as possess more or less diuretic action. Potassium citrate in 10 grain doses, sweet spirits of nitre in 10 or 15 minum doses, potassium acetate in 5 to 10 grain doses may be combined or given separately in water three times a day. Infusion of digitalis in ½ to one tablespoonful doses three times a day or Diuertin in 5 grain tablets. In general to favor absorption the iodides are much in vogue and the syrup of hydriodic acid in teaspoonful doses every four hours, the use of iodide of potassium in 5 to 10 grain doses or the equivalent in sodium or ammonium iodide every four hours dissolved in water or the use of the tincture of iodine U. S. P. in 10 minum doses given in milk every three or four hours are found most reliable. Next to the above the salicylates are much in use. The sodium or ammonium salicylates in 5 to 10 grain doses every three hours. If there is pain and

restlessness, Dover's Powder in 10 grain doses at bed time will be found very advantageous, codeine in small doses or some of the synthetic drugs like veronal, (barbital) trional or sulphonal in 5 grain tablets are also useful.

Surgical Treatment of Pleurisy

The pleural exudate may be removed by forcing air into the pleural cavity and thereby crowding the fluid out, the contained air displacing the exudate. Results have been found most gratifying. The technic is quite simple. A hollow needle is passed between the ribs, 10th intercostal space, into the pleural cavity, and a second needle or better a trocar is now passed into the 8th or 9th intercostal space above connecting the latter with an apparatus by means of which nitrogen gas or filtered air may be forced into the pleural cavity, the pleural fluid being expelled from the lower opening or needle. Before inserting the second needle or the trocar allow as much fluid to flow from the first puncture as will flow out spontaneously, after which use of the air pressure should be made. In most instances nearly all the fluid may in this manner be removed without sudden disturbance of the heart, the inserted air under pressure taking the place of the withdrawn fluid. This also will prevent pleural adhesions or thickening, and the amount of air allowed to enter should equal about ½ the fluid expelled or withdrawn. Restlessness, cough, dyspnea or pain are usually negative. It very much shortens the course of the disease. It is best to connect such an apparatus with a monometer so as to control the amount of air which is to be passed into the chest cavity. Early removal of the fluid followed by the entrance of air or nitrogen is advocated by many, the exudate being usually sero-fibrinous its early removal preventing pleural thickening or adhesions.

Empyema, Rib resection, etc. Scapular line 8 to 10 interspace, posterior axillary line 8th, axillary line 7th or 8th and anterior axillary line 6th and 7th. In empyema the pus may escape in small amounts at a time through the lungs and be expectorated —the process in months may eventually heal out spontaneously. See Chapter 20—"Surgical Treatment of Tuberculosis."

Autoserotherapy. In a number of cases of pleurisy with effusion after the withdrawal of a syringeful of the serous fluid from the pleural cavity and immediately injecting it subcutaneously, the fluid in the chest cavity begins rapidly to diminish, to

be absorbed; in other cases again no result follows this treatment. If the case is favorable a rapid increase in the urinary flow is observed; the tuberculous process, however, is not influenced thereby.

Diaphragmatic Pleurisy. Sacculated Pleurisy. Here the diagnosis often presents many difficulties. The following signs are characteristic of this form of pleurisy, (1) A vertical prolongation from the outer margin of the sternum meeting horizontally along the tenth rib gives at this point a painful spot on pressure, next, the respiratory reflex; quick contraction of the rectus abdominus muscle at the point of attachment on deep inspiration, sometimes a reflex of all the abdominal muscles; (2) Sacculated mediastinal pleurisy may simulate mediastinal tumors or enlarged glands, the characteristic sudden onset and the presence of fever differentiating; (3) Interlobar Saccular pleurisy. Here the diagnosis is often most difficult and a thorough knowledge of the topography of the interlobar fissures is necessary. The accumulated fluid most frequently follows the interlobar septa. Often during life this condition is not recognized when at autopsy a large quantity of pus is found walled off between the lobes of the lungs. If the fluid in amount exceeds 500 cc then the first evidence of disorder generally shows itself on the anterior chest in the anterior axillary line; it is here that the fluid is in closest contact with the parietal pleura, making it easily accessible for exploratory puncture. There may be much pain and pulmonary gangrene may accompany the picture. Interlobar pleurisy may be either dry or moist but in the by far the greater number of instances it is a purulent affair, an interlobar empyema or lung abscess.

Summary and Conclusions

(1) In pleuritis sicca of the apices, immobilization of the arm on the affected side may give quick relief from pain. A tuberculous pleurisy often exerts a most favorable influence on the underlying cause of the disorder and this not only in cases of advanced tuberculosis but in beginning or first stage cases as well. This can not be attributed to a mere mechanical influence; there must be a chemical momentum present.

(2) The muscle pain, spasm or rigidity is diagnostic of apical pleurisy. The trapezeus, pectoralis major, the supra and infra spinatae, even the brachial plexus is sensitive to pressure, and

Sternberg's symptom complex similar to Pottenger's is not reflex as stated but most probably due directly to the inflammatory process in the nerves about the shoulders.

(3) In 90% to 95% of all idiopathic pleurisies in the adult the tuberculous nature is positive and a pleurisy is more or less grave if after withdrawal of the fluid it reappears or accumulates quickly, if there be but little fibrin in the fluid and if it shows a strong tendency to remain localized. Concerning the prognosis as to the future condition, it may be stated that pleuritis is a most favorable manifestation of a tuberculous process. In latent cases generally the process will manifest itself, that is after the pleurisy, on the side opposite, or the side free from pleuritis. In pulmonary tuberculosis of the bases, accompanied by a diaphragmatic pleurisy, the symptoms may simulate a gastric ulcer, such as pains in the stomach, eructation of gases, vomiting, etc.

(4) Remember that pleuritis in an adult is always a very serious question, being generally secondary to a tuberculous process, whereas in the child[2] it is of far less import. It indicates either an already existing active tuberculous disorder or a latent condition; hence every patient suffering from an attack of pleurisy when the physical findings in the chest do not give evidence of active pulmonary disease should be treated as a latent tuberculous individual, be under constant observation, always remembering that very frequently in later years and after the pleurisy has subsided for some time that the lung findings may become positive.

[2]Although pleuritis is frequent in child life, still in the majority of cases the process is arrested, heals out completely, leaving very little if any evidence of the original disorder. In children in but a small minority is pleurisy followed by serious tuberculous disease.

CHAPTER 26

TUBERCULOSIS AND PREGNANCY

General Consideration. The two most dangerous conditions to which women of childbearing age (15 to 45) are commonly exposed are tuberculosis and pregnancy. The mortality statistics from the area of registration, comprising 67.1 per cent of the total population of the United States, shows that 29,200 women between ages of 15 and 45 died in 1915 from the various forms of tuberculosis; 10,134 died from childbirth of which 4,173 were from puerperal sepsis; 5,766 from the various circulatory disturbances; 6,458 from all kinds of digestive disturbances; 5,549 from pneumonia, all types; 5,424 from cancer and other malignant tumors; while for these ages syphilis is given as cause of death 647 times and gonorrhoea 174 times.

Tuberculosis is largely a preventable disease, and most of the deaths from puerperal sepsis and other obstetrical accidents are unnecessary and also preventable.

The campaign of education on the cause and spread of tuberculosis has resulted in a steadily declining death rate from this disease. The dangers of maternity are, on the other hand, calmly ignored by both physician and layman with the assumption that this appalling mortality is the sacrifice which women must lay on the altar of motherhood. The death rate from maternity is just as high, if not higher, today than three score and ten years ago when anesthetics were little used and antisepsis unknown.

Pregnancy with tuberculosis (65) is a most unfortunate and very fatal combination. The cases of tuberculosis with pregnancy are so widely scattered that it has been impossible for any one observer to study and report a series large enough to warrant definite conclusions. Nevertheless, the precentages of the various writers, who have reported small series of cases, correspond so closely that for the present they may be accepted as typical.

1. **Amenorrhoea and Sterility in Tuberculous Women.(181)** Tuberculosis is one of the recognized causes of constitutional amenorrhoea. In a series of 214 tuberculous women in which Norris made a careful study of the menstrual changes, complete

amenorrhoea was present in five per cent and scanty or scanty and irregular flow, in an additional fifty-three per cent.(137) There may be a scanty menstruation or even a complete cessation in the early stages of tuberculosis. In advanced cases menstruation and probably ovulation are often suppressed. There is an old superstition among the laity that amenorrhoea is the cause of consumption and varied attempts are made to induce menstruation in these cases. It is now recognized by the profession, however, that the amenorrhoea is a result of the secondary anemia, and, therefore, one of the body's conservative measures. With the arrest of the tuberculous condition and an improvement in the general health of the patient, there is a return of the menstrual function.

Amenorrhoea is not as suggestive a sign of pregnancy in the tuberculous as in the normal woman; nor is the presence of amenorrhoea without pregnancy a proof of sterility, since ovulation may occur without menstruation. Fortunately, however, tuberculosis, particularly in advanced cachectic cases, may be a cause of sterility.

Women with tuberculosis of the uterus or tubes are usually sterile. Tubal tuberculosis may become latent or even disappear. Carstens,(112) in a recent paper, claims that if nature can remove the myriads of tubercles in the peritoneum, in whatever part they may be, nature will also remove the few that can be found in the tubes, and urges that the latter need not be removed on that account. He reports subsequent pregnancy in two patients whom he had operated for tuberculous peritonitis with tubal involvement. Most of the cases of severe tubal tuberculosis, however, result in a permanent closure of the tubes and sterility.

2. **Incidence of Tuberculosis Among Pregnant Women.** Pregnancy demands an unusual amount of work from the various organs of the body. The relation of pregnancy to the incidence of tuberculosis is debatable, yet it is undoubtedly responsible for the active symptoms of phthisis in a considerable number of patients. Some of these might have shown evidence of the disease without the test of pregnancy, while others would never have developed an active process.

In a recent review of 300 cases together with a review of the literature, Douglass and Harris report as follows: (69)

"In the analysis of the histories of a series of tuberculous women admitted to the Ohio State Sanatorium from December,

1909, to April, 1917, inclusive, it is apparent that in a large number of cases we are dealing with an exacerbation of a latest or quiescent tuberculosis, rather than an initial lesion, and that, in married women, pregnancy is one of the leading factors in lighting up this inactive process."

"Under the term 'pregnancy' we have included the period from conception to the end of the puerperium. To determine its importance as an etiological factor, 392 married tuberculous women were considered, 92 of whom gave no history of pregnancy, leaving 300 cases in the series on which to base our observations. In 88 of these cases, or 29 per cent, pregnancy was the leading factor to which the onset of tuberculosis was attributed. In 24 additional cases, or 8 per cent, pregnancy was a presumptive factor, making a total of 37 per cent."

These figures correspond closely to others found in the literature. The variations, to a considerable extent, are due to differences in methods of making comparisons and tabulations. The following figures are from different reviews. Trembley of Saranac,(179) in a series of 240 cases, had 151, or 63 per cent, who said the tuberculosis originated or, at least, was first discovered after the birth of a child. Turban of Schauta's clinic, reports that 29 per cent of the tuberculous women, observed by him, gave a positive history of the disease originating or becoming definitely recognizable during pregnancy or the puerperium. In a series of 337 tuberculous women reported by P. Jacob and Pannwitz, 25 per cent traced the origin or aggravation of the disease to pregnancy. Scarborough reports 94, or 47 per cent, of 200 married women admitted to the Iowa State Sanatorium in whom the symptoms of active tuberculosis appeared after childbirth. The combined statistics of Funk, Jacob, and Pannwitz, and a number of other observers with a total of over 1000 cases show that 39 per cent believe their infections originated during pregnancy or the puerperium. In view of these reports, it is certain that pregnancy plays an important role in the development of active tuberculosis in women. It must be considered one of the important predisposing causes.

3. **Effect of Tuberculosis on Pregnancy.** There is little indication that spontaneous abortion is any more common in tuberculous women than among others, yet occasionally, tuberculosis is recorded as the cause of the abortion. Any chronic infection may act as a predisposing cause of abortion, but, unfortunately,

only the more severe or hopeless cases of tuberculosis show any considerable tendency to sterility or, in case of pregnancy, to abortion.

Polak and Matthews (181) in their reviews state: "The effect of tuberculosis on the course of pregnancy is practically nil." They quote Emil Sergent as stating that tuberculous women seldom become pregnant, but that if pregnancy does occur, abortion is rare even in the advanced cases with cavity formation. Lobenstine considers it reasonable to believe that abortion is more common in the tuberculous than in the non-tuberculous woman due to the cough, haemoptysis, fever, and the possible tuberculous involvement of the placenta or decidua. It is apparent, however, that mild cases will usually go to term.

4. **Effect of Pregnancy on Tuberculosis.** Tuberculosis may have little effect on pregnancy, but the converse is unfortunately not true. It has already been stated that about 39 per cent of the married tuberculous women believe their active tuberculosis began during or subsequent to a pregnancy. This, undoubtedly, means that pregnancy caused a lighting up of latent and perhaps old lesions in these women.

In cases of known tuberculosis, there may be an increase in the amount of gastric disturbances during the first trimester, anl abortion may result. During the second trimester, a considerable percentage of the patients show a definite improvement due to the increased metabolism, and they may continue to term without any particular difficulty. During the last three months, however, many of the patients lose ground with an alarming rapidity and may die at any stage of labor or the puerperium. For the advanced cases with an exhaustive cough, haemoptysis, and fever, the prognosis is invariably bad. Death may occur at any time during pregnancy, labor, or the puerperium. Most of the advanced cases will have died within a month after delivery.

Existing tuberculosis is usually aggravated by pregnancy. Lobenstine, (184) in the Bulletin of the Lying-In Hospital of New York, states that 38 per cent of their cases were seriously affected by parturition. Lebert believes that 75 per cent of tuberculous women are badly influenced by pregnancy and the puerperium; Diebel, 64 per cent; v. Rosthorn, 70 per cent; v. Bardeleben (180) wrote, from the communications of fourteen physicians, that 71 per cent of tuberculous women grew worse from parturition, and that 47 per cent of the active cases proved fatal

(Lobenstine). Norris and Landis report that in their experience "about 20 per cent of mild, quiescent, pulmonary tuberculosis and 70 per cent of more advanced cases exhibit exacerbations during pregnancy or the puerperium."

It is self-evident that if the tuberculous woman becomes pregnant or the pregnant woman shows signs of tuberculosis, she should have the most careful attention during the entire parturient period, from the beginning of pregnancy to the end of the puerperium. Careful sanatorium care during this period will lessen the dangers to some extent, but the end results are still unsatisfactory. McSweeny and Wang (102) report 18 childbirths at Sea View Hospital, one patient having a second child three years after the first. Ten stated that the onset of their clinical tuberculosis occurred before pregnancy; of these, after labor, four died, three seemed improved, and three seemed unimproved. Three stated that the onset of their clinical tuberculosis occurred at about the time they became pregnant; of these, after labor, one died and two seemed improved. Five stated that the onset of their clinical tuberculosis occurred after they became pregnant; of these, after labor, four seemed improved and one seemed unimproved. The woman with the second pregnancy retrograded during pregnancy and after labor; she was a patient in the hospital when the report was made. In their summary, these authors report that during pregnancy, as near as could be ascertained, ten (55.5%) seemed to retrograde, five (27.7%) to improve, and three (16.6%) were apparently unchanged. After labor, of the moderately advanced cases, seven were improved; of the far advanced, five (45.5%) died, one (9%) improved, and five (45.5%) retrograded. This series while small is valuable owing to the careful treatment afforded.

Polak and Matthews have summarized the effects of pregnancy on tuberculosis as follows:

"Practically all observers agree that labor and the puerperium are the periods of the greatest danger to the woman. During labor, sudden death may occur from cardiac failure, pulmonary oedema, or pulmonary hemorrhage. During the puerperium, the tuberculosis may become fulminating and cause death in a surprisingly short time. Schlimpert, with his great experience in dissection, asserts that the greatest number of deaths from tuberculosis during pregnancy occur during childbed. Accidents during the puerperium are liable to occur in all types of

tuberculosis with active lesions and sometimes of only moderate severity. In other words the puerperium is a period of 'watchful expectancy,' for one can hardly expect to prognosticate correctly in any case, latent or active, where the uncertainty of the reaction is so great."

Bacon (134) estimates that from 1 to 1.5 per cent of all pregnant women have tuberculosis to such a degree that it can be detected if careful examination is made. He quotes Freund as finding 12 cases in 1000 pregnant women examined. Using Bacon's estimate of from 24,000 to 36,000 tuberculous women confined in the United States each year, we would judge from the few carefully observed and reported cases that several thousand of these women will die within a month after delivery. Yet the ordinary tables of vital statistics do not show pregnancy as a contributory cause of death. Until such times as it is so recorded we can have no definite knowledge as to the real mortality resulting from the strain of pregnancy, labor, and the puerperium.

It is generally believed, and quite properly so, that by careful medical attention, with thorough use of absolute rest, feeding, and fresh air, ease of mind, and obedience, many more pregnant tuberculous women would be brought safely through pregnancy and the puerperium with an eventual hope of cure. Bacon advocates this management before and after labor, with a free use of oxygen during labor. Only careful records over a period of years will show to what extent this management is valuable. **It is estimated by Bacon that under present conditions 33 per cent of tuberculous women who become pregnant die in less than a year after delivery.**

5. **Effects of Maternal Tuberculosis on the Fetus.** Tuberculosis (although extremely infrequent) may be transmitted directly from the mother to the fetus **in utero.** This is particularly true in cases of miliary and laryngeal tuberculosis. The tubercle bacillus has never been found in spermatozoon, but Norris believes one may become attached to the spermatozoon and with it reach the ovum[1]. This, however, while possible from the theoretical standpoint, is most improbable. See Chapter 3, The Genitogenetic Route of Infection.

To quote further from Polak and Matthews: "Hauser, in 1898,

[1]Ordinarily the child in utero will not become tuberculously infected; this can only take place if the mother is toxic tuberculous.

found in the literature reports of 18 cases of congenital tuberculosis. Martha Wollstein, in 1905, reported a case of 'proved congenital tuberculosis.' Novak and Ranzal claim that in 70 per cent of the cases of positive tuberculosis in the mother or in the fetus, the placentae contain tubercle bacilli. Schmorl and Giepel assert that in 45 per cent of known tuberculous women the placentae contain tubercule bacilli. Charles Norris states that from a summary of 67 cases of maternal tuberculosis gathered from the literature, 30 per cent presented positive evidence of tubercle bacilli (see Chapter 3) in the placenta. In a personal communication, Norris says that he has found 20 per cent of the placentae of positively tuberculous women to contain tubercle bacilli, and furthermore he believes that it is possible for tubercle bacilli to be transmitted through a normal placenta." These observations are very important, if true. Placentae of tuberculous women should be examined microscopically and treated with antiformin and the results tabulated with a brief of the patient's history. The whole problem of management may hinge on the accuracy of these observations.

The child of a tuberculous mother inherits a tissue weakness and may not have an equal chance with that of the healthy mother. Any chronic disease lowers the mother's vitality and may be expected to interfere to some extent with a healthy development of the fetus. Again, the mother cannot safely nurse these children, and, in most cases, a wet-nurse not being available, they must be raised on a bottle, generally under the same roof or in the same room with a tuberculous mother. Under past and present conditions most of these children will certainly develop some type of tuberculosis. The mortality has been extremely high during the first twelve months. The late Dr. Jacobi (133) estimated that 70 per cent succumb during the first year; Weinberg, 67.9 per cent; Zirkel, 58 per cent; Pankow and Kupferle, 54.5 per cent. Miller and (91) Woodruff working in New York examined 150 children, born of tuberculous parents, and found that 51 per cent were definitely tuberculous, 20 per cent were doubtful, and only 20 per cent showed no signs of tuberculosis. Similar results are reported from Boston by Floyd and Bowditch, who found tuberculosis of the lungs in 36 per cent and infection elsewhere in 30 per cent, or a total of 66 per cent of the children of tuberculous parents with tuberculosis in some form. These results, however, only apply to children who

have remained with their parents. To date there are no statistics showing the degree to which the children may be saved by immediate removal from infected parents.

6. **Nursing and Tuberculosis.** It is generally recognized that it is dangerous for the tuberculous mother to nurse her child. Although it is very desirable for the weak infant of such a mother to have the benefit of mother's milk during the first weeks of life, the dangers from nursing, both from the consideration of the mother and child, are such that it would seem wiser, in most cases, to depend entirely on artificial feeding, if milk from a healthy mother cannot be obtained. This is especially so in the interest of the mother and less that of the child.

For the mother, the puerperium is the period of greatest danger, as at that time particularly, she needs all of her reserve to combat the infection. The quantity and quality of her milk is often impaired. Rarely, the breast may be infected with tuberculosis, but this can only be determined by careful microscopic examination of the breast contents. The child usually contracts tuberculosis from the sputum coughed out by the mother while nursing. This danger may be lessened by careful cleansing of the nipples and having the mother wear a gauze mask.

7. **Birth Control Among the Tuberculous.** Marriage of all persons with active tuberculosis should be prevented. Tuberculosis is second only to gonorrhoea and syphilis as a contraindication to marriage. After the "cure" has endured for a period of two or more years, a man may marry with little danger to himself, his wife, or offspring. But, in view of the great tendency for lighting up of tuberculosis during pregnancy, it is more difficult to determine when the woman may safely marry. Matrimony may interfere with the cure, and pregnancy may soon lead to a fatal termination.

Prevention of pregnancy in the tuberculous is easily advised, but very rarely accomplished. Except in extreme cases it is almost useless to urge abstinence; however, until such times as the woman has gone without any symptoms for a number of years every effort should be made to prevent pregnancy. In the few cases where the abdomen may be opened for other conditions sterilization may, at times, be advisable. The advisability of opening an abdomen for this alone is questionable, and the resection of the tubes **per vaginum** is usually difficult in the nullipara. The so-called operations for temporary sterilization of

women are, in most cases, temporary only in theory.

8. **Prophylaxis of Tuberculosis in Pregnancy.** There is abundant evidence that pregnancy plays an important part in the lighting up of old tuberculous lesions, and it is probable that during pregnancy a woman is more prone to reinfection. Regardless of the debatable question of new or old lesions the danger is real and becomes one more strong argument for carefully supervised care of every woman during the entire period of pregnancy, labor, and the puerperium.

Woman has always paid a fearful price for the old and false belief that childbirth, being a normal function, needs no particular consideration. While under the care of capable obstetricians serious complications are not common, very few women have perfectly normal, physiological pregnancies or labors. Scientific prenatal care, with skilled supervision during labor and the puerperium, will not only increase the number of normal cases, but will also greatly lessen the present high mortality and morbidity.

Tuberculosis is not one of the chief causes of this appalling maternal mortality and morbidity, but with a general improvement in the care of women who are becoming mothers, fewer women will die from tuberculosis. Both the public and the profession must be educated; hospital facilities must be provided; the district or home nursing organizations must be enlarged until they may provide nursing care at a reasonable rate for all women who are to become mothers. These organizations should receive city, county, or state assistance. So far as possible, all tuberculous women should be taken to the hospital or sanatorium for delivery. When beds are scarce, any woman whose condition is good may be taken home in an ambulance at any time after delivery and be cared for by the visiting nurses.

9. **Treatment of Tuberculosis With Pregnancy.** The treatment of tuberculosis complicated by pregnancy will be much the same as when not so complicated. Owing to the dual nature of the condition, however, the obstetrician should always work with the internist.

The tuberculous women, due to her diseased condition, may have an unusual amount of gastric disturbance during the first trimester. It is essential that this be quickly controlled or the resulting loss of strength will greatly lessen the possibility of an ultimate recovery from the tuberculous disorder.

There is undoubtedly a disturbed metabolism with every pregnancy, and the development of symptoms depends, apparently upon the metabolic reaction of the patient and the presence of one or more conditions as possible causes of the nausea and vomiting of pregnancy. The following plan of treatment has given much relief to patients suffering from the gastric disturbances of pregnancy; with slight modifications of diet it may be used for the tuberculous, pregnant women.

1. A carefully taken history will usually reveal any previous gastro-intestinal disturbance, nervous disorder, or other physical condition which makes the patient more susceptible to the metabolic disturbances of pregnancy.

A physical examination may reveal any source of local irritation or infection. Remove any source of infection found in the teeth or in the tonsils. Dental work must not be neglected during pregnancy. Try to correct any uterine displacement. Treat acute cervicitis when present. Any of these may tend to increase the nausea and vomiting of pregnancy, but should be regarded as predisposing rather than causative factors.

2. Keep the patient in bed until the nausea and vomiting is controlled. With the non-tuberculous it may be sufficient to keep the patient in bed for a few hours after breakfast, but the tuberculous need complete rest in bed.

3. Give an antacid, dry type of diet, such as is used in the nervous type of vomiting among the non-pregnant. Solid food may be retained when liquids are vomited. Allow no liquid within the hour before meals or the first one and one-half hours after. The patient may have enough cream or milk to moisten cereals but should have no other liquid. Bulgarian milk may be well borne when sweet milk is not. For the non-tuberculous woman, this diet is high in carbohydrates and low in fat and proteins. But so long as the diet is of the solid type, the percentages of carbohydrates, fats, and proteins may be varied to meet the requirements of the individual patient.

4. Alkalinize the system by the use of alkaline water or sodium bicarbonate. From twenty to sixty grains of sodium bicarbonate, t. i. d. in a full glass of water taken midway between meals and at bedtime may be needed for several days. After the urine becomes alkaline, the dose is reduced to ten grains and continued, t. i. d. If, on subsequent tests, the urine is found strongly acid, the dose of sodium bicarbonate is again

increased. The alkaline treatment is continued throughout pregnancy.

5. Stomach lavage is needed at times. The simplest method is that of having the patient drink several glasses of tepid water, each containing one-third teaspoonful of baking soda. This, when vomited, washes the stomach effectively and causes less irritation than the tube. The tube should be used when stomach tests are indicated.

6. Bromides are of value in the cases not controlled by the dry diet and alkaline treatment. Ten or fifteen grains may be given in a little water, thirty minutes to one hour before the taking of food. The effervescent triple bromide is perhaps best borne by the stomach. Cerium oxalate, two grains every half hour for three doses, is at times useful in cases of simple nausea, but of no value in the more severe. The use of cocaine is not favored, even though good results have been reported.

7. Corpus luteum extract is administered hypodermatically when the history suggests ovarian insufficiency and when there has been one or more miscarriages. It is claimed that both thyroid and corpus luteum extracts increase metabolism, so there might be some benefit from giving it to the tuberculous, pregnant woman.

8. It is believed that prompt management of cases with nausea and vomiting of pregnancy will prevent their reaching the so-called pernicious stage. Patients with the severer forms of vomiting require a special nurse or better be in a sanatorium as it is extremely difficult to secure absolute rest and quiet in the home. If seen before the stage of marked dehydration and severe acidosis, stop all food and liquid by mouth and given glucose solution of from 2 to 5 per cent per rectum. Two hundred fifty cc of the glucose solution containing from thirty to sixty grains each of sodium bromide and sodium bicarbonate may be given every four or six hours as a retention enema, which given with a small tube, is usually easier on the patient than the drip method. It may be advisable to elevate the foot of the bed. As the nervousness is controlled reduce the bromides to thirty grains. After twenty-four or thirty-six hours of this treatment, if the nausea is relieved, the patient may have small amounts of fluid and, later, solid food by mouth.

9. The severe cases seen after marked dehydration is present should have 800 to 1000 cc, N/NaCl solution under the breasts.

When possible, it is advisable to give these patients an 18 or 20 per cent glucose solution intravenously according to the technic of Woodyatt, Wilder and Sansum.(119)

10. Constipation is a common complaint and may be an important factor in causing some of the nausea and vomiting. Various plans of treatment have been tried, but at present milk of magnesia and liquid petrolatum are the two forms of medication most commonly employed. With the alkaline treatment, many patients make a complete recovery from constipation.

Fresh air, rest, properly balanced diet, happy family associations, freedom from household cares, etc., so necessary to every pregnant woman, are even more important to the pregnant tuberculous. It is always better that she have the advantages of sanatorium treatment. A child **in utero** develops, parasite like, by taking from the food and oxygen of the mother and, like parasites, it also takes to the last, regardless of the strength of the mother, and, in cases of disturbed nutrition, the mother suffers out of all proportion to any effect shown by the fetus. Toxins in the maternal blood will certainly affect the fetus, the degree depending upon the nature of the toxins and the age of the fetus. With proper care during pregnancy, the children of tuberculous mothers will, as a rule, be born healthy. Pregnancy is a very serious complication of tuberculosis, and regardless of the treatment, frequently prevents a permanent cure.

10. **Treatment of Pregnancy With Tuberculosis.**[2] In the past, there has been a general sentiment that in case a pregnant woman has an active tuberculosis, she should be aborted

[2]Therapeutic abortion had reached such proportions in Amsterdam and the Netherlands that Tussenbrock (89) engaged in a most critical study in order to ascertain what influence pulmonary tuberculosis had on the mortality of pregnant women. Here at the onset two most important but very difficult questions arose. First, how great quantitatively is the loss in women as a result of pregnancy complicated with tuberculosis? This first question may be answered in three parts, (1) In Amsterdam, the tuberculosis mortality in women during the puerperium, that is, during the first six months after child birth, is very high, but in the following six months is most perceptibly low, so that the average mortality rate for the year does not exceed that of the non-pregnant tuberculous women. (2) In the mortality curve for women after puberty, there is an absence of the sharp lowering which would show a greater mortality in relation to pregnancy and tuberculosis as a cause. (3) In Amsterdam there is only a relatively less frequent mortality amongst the married; this in part must be attributed to other causes than to the process of child bearing. The second question is: have we any correct methods or definite signs by which we may differentiate the pregnant woman who has tuberculosis, but who goes through the puerperium unharmed, from the woman who does not tolerate her pergnancy well and goes all to pieces? This question is still more difficult to answer. Some tuberculous women undoubtedly do not do well under the combined strain of pregnancy and tuberculosis, while other women are not influenced for the worse while pregnant. There probably exists a group of tuberculous women who tolerate pregnancy very poorly; it is, however, difficult to ascertain which class that is, nor do we know what form of tuberculosis so frequently shows a tendency to influence the pregnancy so disasterously. The prevailing opinion, in the Netherlands, that the tuberculosis mortality is greatly increased by pregnancy and the puerperium can not, judging from these observations, be substantiated, and the general belief of an Abortus Provocatus in pregnancy, if accompanied by active pulmonary tuberculous disease, can not be approved. Catharina van Tussenbrock. Amsterdam. Arch. f. Gynack. 1913.—J. R.

if seen during the first three months. The results of interference after the first trimester are so poor that most observers are agreed that if the diagnosis is made after the fourth month she should be carried to term. According to Bacon, the collected results of therapeutic abortion are not favorable, and he does not believe abortion justifiable in over 10 per cent of the pregnant tuberculous women. Veit (182) found that there was no improvement following abortion, in 43 per cent of the cases collected by him. Trembley, at Saranac Lake, with patients under most favorable conditions, has not seen enough improvement after abortion to warrant the establishment of a general rule. v. Bardeleben states that 50 per cent of his cases died after the pregnancy had been terminated by therapeutic abortion. It is impossible to estimate the percentage of aborted women who would have died had the pregnancy continued. A study of the literature leads to the suspicion that the end results would be about the same without abortion if the patients could have adequate care during the entire period of pregnancy, labor, and the puerperium. All cases of therapeutic abortion should be reportable to the health department, and the end results carefully tabulated for future guidance.

Dr. Bacon says of therapeutic abortion (59):

"It certainly should not be the general rule, because of several reasons. One is this: the results of abortion are not very good. There are two indications—prophylactic and vital—for abortion to prevent further trouble, and save the woman's life. The vital indication is worthless. If the woman is in such condition that she is pretty sure to die if she goes on with the pregnancy, she will be pretty sure to die after abortion is induced, so that it is not really a reasonable indication. Possibly we want to save hei to her family a little longer, but most authorities agree that women go down pretty nearly as fast after the operation as without it."

"The prophylactic indication is the prevention of further increase of trouble in a woman that has a good hope of success in her fight with tuberculosis. Before deciding on this indication, it is necessary to answer the questions: How can the pregnancy be managed? Can the patient be treated in her home or in a sanatorium properly? If she is sent to a sanatorium to be cared for in an ideal way during her pregnancy, and the labor conducted in a proper way, the child taken from

her and properly cared for, if that can be done, I believe it is very much better than induction of abortion. I should say that it is very seldom that induction of abortion is indicated. I think it will grow less and less as we can improve our management."

The tendency to abort all proven active cases of tuberculosis, when possible, during the first three months of pregnancy is largely the result of the old belief that the child was usually tuberculous. Should not the present knowledge that the child, when removed from direct exposure following birth, will be healthy, as a rule, lead to a greater consideration of the child?

EDUCATE THE PEOPLE TO THE DANGER OF PREGNANCY IN THE TUBERCULOUS. PREVENT THE MARRIAGE OF THE PEOPLE WITH ACTIVE TUBERCULOUS LESIONS. LESSEN THE DANGERS OF LIGHTING UP OLD LESIONS DURING PREGNANCY BY SCIENTIFIC PRENATAL CARE. LIMIT THE THERAPEUTIC ABORTION TO THE COMPLICATED CASE WHERE THE CHANCES OF A HEALTHY CHILD ARE PRACTICALLY NIL AND THE PREGNANCY MAKES THE PROGNOSIS HOPELESS FOR THE WOMAN. REQUIRE THAT ABORTED CASES BE FOLLOWED UP AND THE END RESULTS REPORTED FOR FUTURE GUIDANCE.

In the few cases where early termination of pregnancy is believed advisable after unbiased consultation of experienced observers, the method of emptying the uterus should be left to the judgment of the operator. If the case is seen within the first two months, the cervix may be dilated with the Hegar dilators and the ovum removed with the placenta or curette forceps. After the first two months, the vaginal hysterotomy is perhaps the best procedure. Frankenthal believes in the slow dilatation of the cervix with tents. Other methods, as the use of a catheter or pack, are less satisfactory and perhaps more dangerous. When possible these operations should be accomplished with local anesthesia. Both ether and chloroform are believed to be dangerous to the tuberculous.

Radical operations to insure permanent sterility have been advocated, but are not favored as they will increase the immediate mortality. The legality of such a procedure may also be

questioned in the absence of local pathology justifying the removal or mutilation of the organs of generation.

The various writers on tuberculosis and pregnancy are apparently agreed that, unless terminated within the first three months, the pregnancy should be carried to viability or term. Some would induce premature labor on the ground that the mother's chances are bettered. This would hardly seem justifiable as it lessens the chances of the child without materially improving the prognosis for the mother. It is believed that most women should be carried to term and the labor made as easy as possible with the use of analgesics and usually low forceps for all women with normal conditions of the perineum. The expulsive contractions of the second stage may be responsible for grave circulatory disturbances. Pituitrin should not be used as a substitute for forceps in these cases.

Hypodermic medication with heroin, morphin, or morphin and scopolamin may be used to control a painful first stage, and the use of nitrous oxid-oxygen analgesia for delivery. A gauze filter may be placed in the inhaler so as to prevent contamination of the tubes and gas bags. The inhaler should always be carefully cleansed with soap and water and sterilized with alcohol or lysol solution. It is advisable to wash the tube and bags with running hot water. C. H. D.

CHAPTER 27

TUBERCULOSIS AND PULMONARY HEMORRHAGE

Hemorrhage in Tuberculosis. Pulmonary Hemorrhage. Bleeding from the Lungs. Symptoms of Pulmonary Hemorrhage. Methods for Treating and Controlling Hemorrhages.

General Consideration. If an individual who for some time has been the victim of a troublesome cough, in a fit of coughing brings up a mouthful of blood that in all probability came from the lungs, and if at the same time he is suffering from active pulmonary disease, then the hemorrhage was positively pulmonary. Most all pulmonary hemorrhages are of a tuberculous nature. In about 20% of all cases it is an initial symptom but in the greater number (80%) it is a terminal event. In old chronic cases of pulmonary tuberculosis a little pulmonary oozing, a small hemoptysis is of very slight importance and does not call for immediate treatment. This slight hemorrhage is simply the expression of a favorably prognostic sign showing that the tuberculous process is favoring a contraction.

A pulmonary hemorrhage usually comes on suddenly without any previous symptoms or warnings; however, occasionally patients complain for days of premonitory signs of a dull heavy feeling, pressure like symptoms. This is particularly mentioned by those who have had repeated hemoptysis. Meteorological conditions seem to influence pulmonary hemorrhage. Pulmonary hemorrhages are more frequent on days of greater humidity than on the dry. There seems to be some connection between pulmonary hemorrhage and moist days, as if moisture in the air would play an important role; air pressure and the air currents also are accessory factors. The bleeding is less frequent with the north and east winds and more so when the winds are from the south and west, perhaps because the south and west winds carry more moisture. It is twice as frequent in men as in women. Hemorrhage appears more frequently as a recurring attack while in a high altitude and is much less frequent in the moderately high or in the low lands. A pulmonary hemorrhage

means at least a teaspoonful of blood—a little blood streaked sputum should not be considered as a hemorrhage. In considering pulmonary hemorrhages we must be sure that the blood was coughed up, not vomited, that it did not come from the stomach, from an oesophageal varix nor from the mouth or the posterior nares.

The Treatment of Pulmonary Hemorrhage (a) Mechanical; (b) Medicinal

Any treatment, either mechanical, medicinal or otherwise, has for its prime object the bringing about of an increase in the coagulability of the blood.

(a) **By mechanical methods.** The coagulability of the blood may be increased and the intrathoracic pressure decreased by the old method of applying a ligature very tightly about one or both lower extremities, usually around the middle of the thigh, allowing the constriction to remain from a half to three-quarters of an hour, after which the binder must be slowly and gradually loosened. This by slowing the return circulation of blood to the chest cavity, produces venous stasis with increased carbonization of the blood, increased viscosity and consequently increased coagulability. Another very simple and efficient method for controlling pulmonary hemorrhage is by means of adhesive straps applied firmly about that half of the chest cavity from which the hemoptysis is supposed to come in the manner so much in use in pleurisy and intercostal neuralgia.

(b) **By medicinal means.** The coagulability of the blood may be brought about by remedies which will first increase the fibrinogen content of the blood and secondly by such medicaments as will tend to increase the rapidity of the blood's coagulability, or both. The fibrinogen may be greatly increased by injecting subcutaneously from 10 to 20 cc of some body foreign albumen, for which purpose horse serum is now most suitable, as well as available. This injection is followed by an increase of the fibrin ferment, globulin, and the fibrinogen coagulability is increased 10 fold within 24 hours, remaining at that point 5 to 8 days and then again gradually returning to normal. In place of horse serum a sterilized gelatin solution may be employed; from 30 to 60 cc of a 2 per cent solution may be given subcutaneously and like all solutions intended for subcutaneous medication must be prepared with all aseptic precautions. This gelatin solution has a decided advantage over horse serum in not producing an

anaphylactic reaction which must always be considered when using the latter.

To increase the rapidity of the blood's coagulability remedies are used which will produce, either local or general, a change in the condition of the blood in the sense of bringing about a dilution so that the blood vessels absorb more lymph fluid making the blood more hydrolytic. Experience has demonstrated that such blood possesses much greater and quicker properties to coagulate and R. van den Velden has shown that nothing favors this more than an intravenous injection of about 5 cc of a 10 per cent sterile salt solution. By this method the increased coagulability reaches its maximum in about 20 minutes, dropping again back to normal in about one hour when the injection may be repeated. To avoid these oft-repeated intravenous injections which to some patients may be very irritating, a subcutaneous one may be given, using from 100 to 200 cc of a 2 per cent sterile salt solution by the slow or drop method. Pulmonary hemorrhage may also be controlled by remedies which will bring about a change in the size of the lumen of the bleeding vessel. To bring about this change only one remedy is known in medicine and that is Adrenalin, but this remedy will also raise the blood pressure, which in consequence thereof may be followed by still more serious difficulties, as a thrombus may be loosened and in this may become the source of a renewed hemorrhage. This remedy in about one or two milligram doses may be given either by mouth or may be subcutaneously administered.

A remedy used to lessen the quantity of blood flowing from a bleeding vessel in a given time is Amyl Nitrite, but it is unreliable and not suitable because its therapeutic effect is of too short a duration. Two other remedies which are of very much importance in the treatment of pulmonary hemorrhage are tincture digitalis and tincture aconite root, depending upon each individual case as to which one is to be given. In the presence of cardiac weakness and low blood pressure, feeble, hardly palpable pulse, and repeated small hemorrhages pointing to a passive congestion, give tincture digitalis, but if the pulse is full and bounding, much elevation of temperature, much excitement, slightly high blood pressure and much bleeding as is most frequent in pulmonary hemorrhages, then use tincture aconite. Of other remedies of undoubted service in pulmonary hemorrhage are potassium bromide in 20 to 30 grain doses every three hours,

dissolved in cold water and calcium chloride or lactate in 5 grain doses every three hours. Remedies like acidum tannicum, acidum gallicum, plumbum aceticum, ferri chlorida hydratis canadensis and the newer ones like Stypticine, Styptol, etc., should be discarded. Fluid Extract Ergot is of no value in pulmonary hemorrhages; it is positively contraindicated, for in order to produce the necessary contractions of a blood vessel to check the hemorrhage it would be necessary to give it in enormously large, in toxic, doses.

In recent years the use of substances derived from tissues and organs of animals, chiefly colloidal in character, has found great therapeutic favor in the treatment of pulmonary hemorrhage, in some quarters. Organotherapy or opotherapy as it is known (from opos. juice) is applied today in the sense of supplying animal derivatives to a diseased human organism with the desire to give either to such a diseased body something in which it usually is deficient, a condition which is chiefly manifested in body weakness, that is something to give strength, or to lessen the amount of labor required in the human economy by supplying the necessary protein already prepared and requiring simply absorption by the body tissues without any elaboration. It is also given with a view of stimulating the secretory functions in a diseased body, as they are usually in abeyance.

For the treatment of pulmonary hemorrhage, Opotherapy Pulmonalae, or ingestion of dried, sterile, powdered lung extract has not been proven to be of any service, but Opotherapy Hepatica has been found to be of undoubted value. Gilbert and Carnot have found the giving of the dried, powdered, sterilized liver extract to be a sovereign remedy in controlling pulmonary hemorrhage. The Extractum Hepatica is given in pill form of about 5 grains each, giving from 5 to 8 pills daily. These investigators recommend the giving of calcium chloride with extract of opium, simultaneously administered, but find that all other remedies are of very little avail when compared to liver extract. Trousseau recommends the use of powdered ipecac root in doses sufficient to produce nausea should Extractum Hepatica not prove promptly efficacious. In place of ipecac root the isolated alkaloid Emetin offered in ampules in proper dosage is now used by many clinicians. Rest has tested the action of the extract prepared from the posterior lobe of the pituitary body in 12 cases of most profuse pulmonary hemorrhage which were not influenced

by the ordinary methods of medication, with the most gratifying results; in most all cases the hemorrhage stopped quickly and promptly.

In repeated and profuse hemorrhages occurring at longer or shorter intervals in which the usual medicinal treatment is of no avail, the establishing of an artificial pneumothorax may be most desirable. By proper compression of the lungs, bringing the cavity walls into closer apposition, the vessels from which these repeated hemorrhages occur are closed, and a permanent arrest of the hemorrhage may confidently be expected if the pneumothorax is maintained for a sufficient length of time.

Prophylaxis

At the present time with our ever-increasing exactness, our knowledge of the use of medicines, of the etiology and causative factors of most diseases, with a closer observation and surveillance of patients at sanatoria as well as the ambulatory cases, we are in a position in many instances positively to anticipate a pulmonary hemorrhage and in many such cases are able by proper means and methods to avert it. In an active tuberculous subject a sudden rise in blood pressure may be the forerunner of a pulmonary hemorrhage. This rising phase of hypertension may gradually lead to hemoptysis or it may continue for weeks before the actual hemorrhage occurs, and in many tuberculous individuals with a tendency to bleeding this hypertension will be found constant with a gradual increase in tension up to the moment of the hemoptysis; hence, frequent blood pressure observations may point out the prophylactic treatment necessary in many cases of threatened hemorrhage, as the blood pressure in the tuberculous is usually very low, any tendency to a gradual increase of the tension without a corresponding improvement in the patient's condition should always be received with much scepticism and immediate rest instituted.

Another method intended as a protective measure applied to patients that have had a previous pulmonary hemorrhage is by administering autogenous vaccines. Judging from the literature on this point, the use of these vaccines of mixed infection is a favorable factor in preventing hemoptysis in proportion of 1 to 3. The theory that the administration of autogenous vaccines to the tuberculous subject may inhibit further hemorrhages is based on the fact that the vaccines of mixed infection stimulate the antibody productions, preventing further action of the invading

organisms upon the weakened, damaged and constantly destroyed lung tissue, protecting and strengthening the structure of the lung surrounding the blood vessel, thereby preventing further bleeding.

Practical Rules for Controlling Pulmonary Hemorrhage

In a given case of pulmonary hemorrhage the following general directions will be found both serviceable and practical.

Put the patient at once into a comfortable bed with the head and shoulders slightly elevated. Instruct the attendant or nurse that the sufferer must not be moved or turned until all signs of hemorrhage have been for some days arrested. Exclude all idle gossipers and visitors from the room. Keep the room well ventilated, somewhat darkened. As soon as the patient is in a comfortable position administer a teaspoonful of sodium chloride or salt dissolved in a glass of cold water; it is at this point that the inhalation of Nitrite of Amyl may be of service. Apply an ice bag over the heart and give by mouth oft-repeated small pieces of ice, which the patient should chew, swallowing the still undissolved particles. As it is also very necessary to limit the intake of liquids, especially water, in order to lessen the amount of fluid in the pulmonary circuit as well as to maintain a comparatively low blood pressure, the giving of ice in this manner will serve a double purpose.

To quiet the nervous fears and to slow the rapid action of the heart, a hypodermic injection of morphine 1/4 grain, and 1/150 atropine, should be given. At this time no attempt should be made by the attending physician at a thorough examination of the chest; light palpation as well as auscultation may be allowable, but percussion must be strictly withheld; a closer examination is permissible only after a few days. All drinks and foods must be given cold; solid foods are prohibited. Warm drinks and all stimulating beverages are strictly interdicted. The following prescriptions should be given alternately in cold water every 1 1/2 to 2 hours:

Prescription No. 1.

Take: Tincture Aconite root, M. xxiv.....................(1.5 cc)
Tincture Opii Deodorated, drachms two.............(8.0 cc)
Antipyrene, drachms two ....(8.0 gms.)
Syr. Orange Flowers, ounce one..................(30. cc)
Aqua Cinnamon, sufficient to make three ounces..(q.s.ad. 90.0 cc)

Directions: One teaspoonful in little cold water once in 3 or 4 hours.

Prescription No. 2.

Take: Sodium Bromide, drachms six....................(24.0 gms.)
Calcium Chloride or Lactate, drachms two.........(8.0 gms.)
Syrup of Licorice root, ounce one.................(30.0 cc)
Aqua Peppermint, sufficient to make three ounces (q.s.ad. 90.0 cc)

Directions: One teaspoonful in a mouthful water once in 3 or 4 hours.

These two prescriptions (No. 1 and 2) should be given alternately once every one and one-half to two hours. Here in the second prescription licorice syrup is added in order to differentiate the two solutions.

This at least in a measure will check the immediately dangerous hemorrhage. Should the hemorrhage still continue in spite of all, the intravenous injection of 5 cc of a 10 per cent solution of salt should be given or 100 or 200 cc of a 2 per cent salt solution subcutaneously administered, or a pint or more of Normal Salt solution may be given by rectum (retention enema) according to the usually employed drop method. Ligatures applied to the extremities should be used earlier. Injections of horse serum 10 to 20 cc or gelatin 30 to 60 cc of a 2 per cent solution may be resorted to if the case is not benefited by the simpler methods, leaving the use of the other recommended remedies as well as artificial pneumothorax to the discretion of the physician in attendance.

CHAPTER 28

TUBERCULOUS LARYNGITIS

Tuberculosis of the Larynx. Laryngeal Tuberculosis. Laryngitis Tuberculosa. Laryngeal Phthisis. Throat Consumption.

General Consideration. By way of introduction to the subject of laryngeal tuberculosis, and in order to indicate its relationship especially to pulmonary tuberculosis, it will be well at the outset to make certain statements of facts: Laryngeal tuberculosis, of itself, is an exceedingly serious disease; it does not exist unassociated with pulmonary tuberculosis for more than a short time, if it exists independently at all; laryngeal tuberculosis is a very common complication of the pulmonary form, and affects profoundly and unfavorably the prognosis of that disease. It is prone to produce very severe pain, and by reason of this pain, induced by swallowing, breaks down one of the most important bulwarks of resistance to both the pulmonic and the laryngeal disease, namely, the taking in of nutriment. (27) (6).

Etiology. In approaching more closely the subject of (57) laryngeal tuberculosis, let us attempt to understand the anatomic and physiologic facts underlying the production of tuberculosis in the larynx. In probably much more than ninety per cent of the cases of tuberculosis of the upper air passages, the disease implants itself, not in the mouth, nose, pharynx, or trachea, but in the larynx. What is the explanation of this fact, that out of the perhaps fourteen or fifteen inches of distance between the lungs and the outer air, this little space of two inches, or less, should suffer so vast a preponderance of vulnerability to the tubercle bacillus? The larynx constitutes for the purpose of this subject simply a peculiar anatomic and physiologic section in the passages through which all air and secretion must pass between the outer air and the lungs. But that statement applies as well to the trachea, below, and to the pharynx, the mouth, and the nose above, all of which spaces are several times more roomy than is the larynx. If we are to find the explanation for the extraordinary susceptibility of the larynx to tuberculosis, compared to the

other upper air spaces, we evidently must examine the peculiarities of the larynx itself.

Two anatomic facts are significant. The larynx is not only extremely irregular in its anatomical conformation, but it is also exceedingly narrow. In passing through this narrow, irregular laryngeal space, it is easy for the air, dust, or secretion, whether moving upwards or downwards, to impinge on the epiglottis,

Fig. 45. The normal larynx. Act of phonation (viewed from above)

1. Vocal cord (true cord).
2. Ventricular band (false cord).
3. Epiglottis.
4. Glosso-epiglottic ligament.
5. Cushion of the epiglottis.
6. Ventricle of the larynx (Morgagni).
7. Ary-epiglottic fold.
8. The cartilage of Wrisberg.
9. Rings of the trachea.
10. Inter-arytenoid fold (commissure).
11. Posterior pharyngeal wall.
12. The cartilage of Santorini.
13. Pyriform, or pyramidal sinus.
14. Bulbus portion of the aryteno-epiglottidean fold.

the false cords, the true cords or to lodge in the sinus of Morgagni or the subglottic space.

Not only is the larynx narrow and irregular, but it is one of the two or three most active organs in the body. It moves quickly and intensively in talking, both with respect to the inner (intrinsic) and the outer (extrinsic) musculature. In this movement of talking not only is the mucous membrane, especially in the arytenoid region, involved, but the vocal cords themselves are brought repeatedly into contact, especially if conditions of

inflammation be present. The larynx also moves in coughing. In this process, the patient inhales deeply, the vocal cords close tightly together, the musculature of the chest wall contracts on the air in the lungs, thus producing air tension against the closed vocal cords, the cords open, and the air is projected violently upwards.

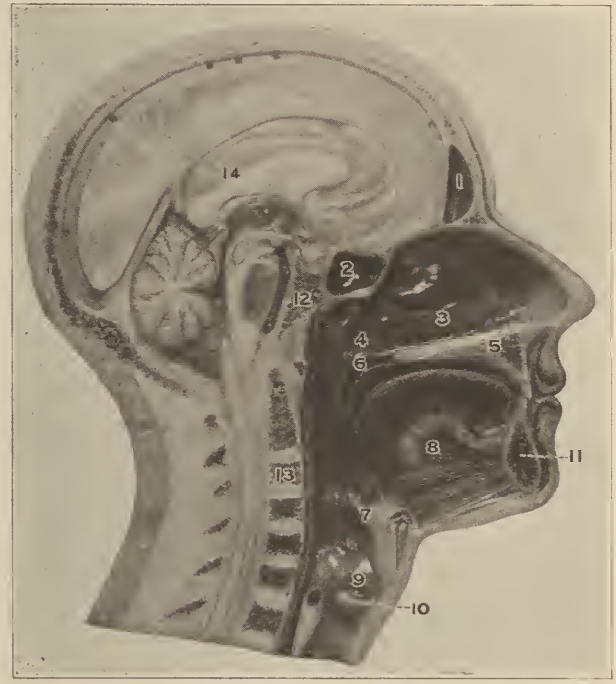

Fig. 46. Sagittal section of the upper air passages.
A. and P. Seifert, Berlin
(From wax model, E. L. Kenyon collection)

1. Frontal sinus.
2. Sphenoidal sinus.
3. Lower turbinate body.
4. Orifice of Eustachian tube.
5. Hard palate; superior maxillary bone.
6. Soft palate.
7. Epiglottis.
8. Tongue; note the intimate muscular relationship between the larynx and base of the tongue.
9. False vocal cord.
10. Vocal cord.
11. Lower maxilla.
12. Base of sphenoid.
13. Body of spinal vertebra.
14. Brain.

There is also movement of the larynx in swallowing. Talking and coughing effect especially the arytenoid and vocal cord re-

gions. Swallowing affects especially the upper aperture of the larynx, and the epiglottis. In swallowing, the bolus of food is received on the upper surface of the tongue and the tongue forces it backwards to the pharyngeal wall; the soft palate raises, the entire larynx is strongly elevated against the epiglottis, the epiglottis against the hyoid bone and base of the tongue. The base of the tongue moves backward and downward, and meets strongly the upmoving larynx, while the bolus is grasped by the oesophageal peristalsis. Thus we have a picture of the under-

Fig. 47. Interior surface of left side of larynx
(From wax model, E. L. Kenyon collection)

1. Trachea, showing cartilaginous rings.
2. Junction of trachea and larynx.
3. Subglottic space of larynx.
4. Left vocal cord, as if in the act of phonation.
5. Sinus of Margagni.
6. Left false vocal cord.
7. Epiglottis.
8. Thyroid cartilage.
9. Cricoid cartilage.

lying anatomical and physiological conditions which constitute the foundation for the lodgment of the tubercle bacillus in the larynx.

It becomes necessary to show what factors encourage the lodgment of the tubercule bacillus. The bacillus gains access to the laryngeal structures either (1) by penetrating the superficial layers of the mucous membrane; (2) by way of the blood; or (3) by way of the lymphatics. While either of the two last sources of infection are certainly possible and, theoretically, may occur, it is believed that the larynx is almost always infected by penetration of the mucous membrane from the lumen of the larynx. One sometimes has the opportunity of witnessing in the pulmonary patient the invasion of a wound in the mouth, as after tooth extraction, by the tubercle bacillus.[1]

Long ago Wm. E. Casselberry pointed out how inflammatory infiltration tended to encourage minute cracks in the laryngeal mucous membrane, especially where the arytenoid mucous membrane joins the posterior wall, and he also pointed out how the movements of the arytenoid in talking encouraged these abrasions.

Chronic laryngitis (non-tuberculous) has been mentioned by some authors as an important preliminary preparation for the local lodgment of the tubercle bacillus. That simple chronic laryngitis is extremely important in this connection does not seem to be clear. However, in so far as pharyngeal abnormalities, for example, pharyngitis, nasal accessory sinus disease, or other nasal abnormalities, constitute factors underlying a chronic laryngitis in a patient, the eradication of such abnormalities is to be given consideration. Another possible etiological factor lies in the lingual tonsil. If large, it undoubtedly may be responsible for irritation in the larynx from mechanical causes, as it lies right above the epiglottis, on the base of the tongue. If in such a location it contained actual infection, the products of that infection would certainly tend to get into the larynx.

A most striking picture of resistance of the mucous membrane of the larynx to the tubercle bacillus is witnessed in a large percentage of patients with pulmonary tuberculosis. For months the surface may be continuously bathed with infectious secretion from the lungs without local implantation of the disease. Finally, however, if the pulmonic disease becomes progressively more and more serious, the larynx, in the course of the downward progress of the patient's resistance, usually succumbs.

Since invasion of the laryngeal mucous membrane occurs under such varying pulmonic conditions, relatively accidental factors may often determine the invading process. These factors concern both constitutional and local resistance. Constitutional resistance diminishes as the pulmonic disease progresses. So also does local resistance, until in the final stages of pulmonary tuberculosis, the larynx becomes involved in nearly all cases (probably more than 75%). When the constitutional resistance is relatively high, as in the early stages of a slowly progressing pulmonary tuberculosis, the larynx should not become involved, unless through diminished local resistance. In an organ so narrow, so irregular, and so movable as the larynx, which is continuously exposed to tuberculous infection, it is evidently of the greatest importance that the elasticity of the mucous membrane should not be impaired.

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[1]See Chapter XXXIII—"Tuberculosis of the Eye and the Mucous Surfaces"—See Footnote on page 448.—J. R.

It is common for the pulmonary patient to locate the source of his cough in the vicinity of the larynx which signifies that the secretion is being driven upwards until it meets with laryngeal obstruction and there it sticks, irritates, and produces a cough. Oedematous infiltration is one of the consequences; impaired elasticity is another, impairment of the epithelial resistance of the mucous membrane, and even cracking of the surface, are then easy possibilities. The tubercle bacillus already present in the larynx, is, through the agency of cough and other laryngeal movement factors, presumably **churned** into a more or less pre-injured mucous membrane.

Primary Tuberculosis of the Larynx (163). Very much has been said in support and denial of the proposition that laryngeal tuberculosis may precede tuberculosis of the lungs. No theoretic reason exists which excludes the possibility of primary laryngeal tuberculosis, but this is probably a rare occurrence. Sir St. Clair Thompson believes that tuberculosis of the larynx can be primary, and cites one observed case in which the laryngeal disease preceded any sign elswhere for three full years.

The great difficulty is in proving the priority of the laryngeal invasion. When laryngeal tuberculosis is discovered as the first evidence of tuberculous disease in the body, the diagnosis of pulmonary disease practically always follows. Usually, if a laryngeal tuberculosis exists as a supposedly primary disease an old latent tuberculous focus in the lung has probably pre-existed for some time; however, laryngeal tuberculosis may be primary and failure to prove pulmonary tuberculosis at the time is not sufficient reason for denying the truth of the laryngeal diagnosis.

Course of the Disease. This varies with the clinical picture, i. e., whether the pulmonary or the laryngeal disorder predominates. Most frequently the laryngeal disease follows late to the pulmonary form and usually shortly before the end. As stated, in most instances pulmonary tuberculosis is the chief difficulty, laryngeal generally secondary, and only exceptionally does the laryngeal disease play the leading part.

Physical Manifestations of Laryngeal Tuberculosis. Visual study of the larynx is technically difficult, and, relatively to our study of the skin, the mouth or nasal organs, necessarily inaccurate. Not only is the organ small, fixedly distant from the eye, and imperfectly lighted, but it must usually be studied as depicted in a small throat mirror image and always from one

point of vantage only. This is without consideration of the additional difficulties resulting from sensitiveness, and consequent hindering movements, of the patient. If the study of this organ could be carried out with the accuracy with which dermatological lesions may always be studied, the earlier manifestations of any laryngeal disease could be as well defined as are those of the skin.

The earliest manifestations of laryngeal tuberculosis are, for the above reasons, not even yet accurately defined and their recognition is dependent upon long and extensive laryngeal experience. They resolve themselves into (A) general paleness of the mucous membrane; (B) localized discoloration and infiltration; (C) localized ulceration.

(A) The paleness of the mucous membrane reflects the general anemia of the patient and is to be expected in this disease. It is, however, offset many times by actual hyperemia, and especially by a peculiar dusky redness, dependent, for example, upon persistent breathing of cold air in winter, a simple laryngitis, or, the rarely excellent general condition of the patient.

(B) Localized discoloration and infiltration gains its significance largely by its isolation and posterior location. The favorite sites are posterior, often at the junction of one vocal cord with its arytenoid, or in the posterior commissure at the side of the arytenoid, or on the laryngeal surface of the epiglottis. They are small, usually dusky red, and but little elevated and their significance is enhanced by their slowness of growth and great persistence.

(C) Ulceration may inaugurate the tuberculous process. The locations are apt to correspond with those just mentioned for localized infiltrations. The ulcers when first seen may be very small, shallow, irregular, and covered by a thin greyish-white secretion. General paleness of the mucous membrane always adds to the diagnostic significance of any lesion.

When such beginning lesions progress and especially if others be added, the picture of the larynx may attain striking variety and irregularity of disease manifestation. Understandable description is beyond possibility. The lesions considered separately resolve themselves into (1) infiltration; (2) tumor-like masses; (3) ulceration; (4) miliary protuberances. Eventually in essentially all progressive cases ulceration sooner or later su-

pervenes, but its advent may be relatively delayed. Any lesion may undergo ulceration.

(1) **Infiltration.** Infiltration may involve one, or later, both vocal cords. The cords then appear rough and irregularly enlarged. Between the arytenoids (the posterior commissure), in-

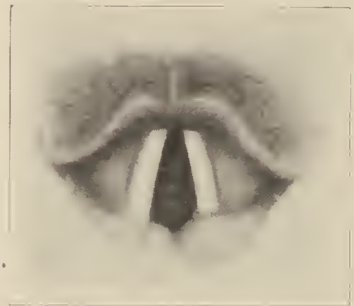

Fig. 48. Tuberculous Laryngitis. Swelling of the Arytenoid.
E. Fletcher Ingals Collection, Rush Medical College

filtration (thickening) may occur, even in pulmonary tuberculosis, without local tuberculous significance, resulting apparently from excessive coughing and irritation. The same may be said of bilateral enlargement of the aryteno-epiglottic folds. If the latter enlargement be asymmetrical, and if the typical pear shape

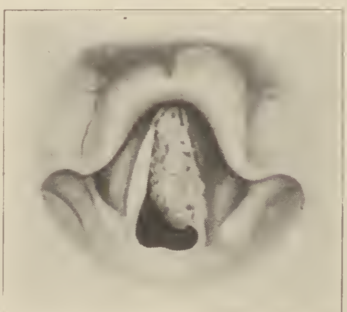

Fig. 49. Tuberculous Laryngitis. Large Granulation Tumor of the Left Vocal Cord with Congestion of the Epiglottis
E. Fletcher Ingals Collection, Rush Medical College

be present, even when bilateral, the picture becomes almost pathognomonic. The epiglottis is also commonly subject to infiltration and this may be marked.

(2) **Tumor Formation.** Tumor-like bodies are common especially in the later manifestations, but may also begin the local

disease. They may have smooth surfaces, appear compact and solid, or they may be irregular, soft, pale, and fungous, or papillomatous. These bodies may be surrounded by ulcerated surfaces, or themselves be ulcerated. They may be small, or they may almost occlude the lumen of the larynx. The site of growth is most often posterior, especially about the arytenoids and posterior commissure. As the disease progresses, increasing ulceration complicates and adds irregularity to the picture.

(3) **Ulceration.** The marked tendency to ulceration is the most serious aspect of laryngeal tuberculosis. It often constitutes the very first indication of the disease, it becomes more serious as the disease progresses, and is the chief cause for the characteristic pain of the laryngeal sufferer. The ulceration,

Fig. 50. Tuberculous Laryngitis. Superficial Ulceration. Fungous Granulation of Posterior Commissure
E. Fletcher Ingals Collection, Rush Medical College

when typically manifested, is shallow, with irregular "worm eaten" edges, irregular floor, and thin greyish-white secretion. A tendency to breaking down into ulceration in spots is characteristic, small ulcers existing between islets of intact mucous membrane. At times, especially on the posterior wall, the perichondrium and the cartilage itself is invaded with this spotted ulceration. The cartilage may be invaded by the tuberculous disease without ulceration, and this is likely to be accompanied by much infiltration.

The epiglottis is frequently ulcerated both superficially in the mucous membrane, and deeply in the cartilage, and any portion, or all, of the organ may thus become destroyed. The surface of one, or both, vocal cords may be more or less extensively ulcerated, and, often, the irregularity of the destructive effects produces a typical saw-edged appearance of the cord. Infiltration,

different types of tumor formation and ulceration, may be present all in the same patient, and as has been said, the infiltrated and tumor areas may themselves be ulcerated.

(4) **Miliary Form.** Another manner of typical appearance is also sometimes presented by the presence of scattered small millet seed sized rounded, greyish, or yellowish, elevations of disease in the mucous membrane. Such deposits of miliary tubercles may also be seen in generalized miliary tuberculosis.

Pathology. The microscopic pathology is not essentially different from that of tuberculosis in other parts of the body. See Chapter 8, page 56 "Histology and Pathology."

Symptoms. Cough, irritation, impairment of the voice, localized pain, difficulty of swallowing, and, rarely, dyspnea due to laryngeal stenosis, are the localized symptoms of the disease.

In any manner of laryngeal inflammation the most evident symptom is a local irritation that produces cough. Since, however, cough, due to the lung disease is present, the local lesion may go unrealized by the patient, and especially the physician, until it produces other and more distinctly localizing symptoms, particularly hoarseness and laryngeal pain; and such delay in recognition is not uncommonly fatal to the patient. This fact emphasizes the importance of systematic laryngeal examination in all cases of pulmonary tuberculosis. On the other hand, if the primary symptoms referable to the larynx are those produced by laryngeal tuberculosis, the beginning localizing symptoms may be irritation with cough, or hoarseness alone, or both combined.

Laryngeal tuberculosis may be present for some time without pain. When pain does occur, it is the most distressing symptom of the condition and may be of any degree and so excruciating does the pain often become on swallowing, that the patient would actually rather starve than go through the act of taking nourishment. Under such circumstances, the patient is steadily directed towards his final end as he is no longer willing to take the nourishment upon which his resistance to the disease depends.

In certain conditions, even in the absence of ulceration, movement that involves laryngeal infiltrations, especially in the region of the arytenoids may result in a moderate degree of pain. But the more serious degrees of pain are dependent upon sensitive ulcerative surfaces, with their nerve endings, exposed to rubbing and to pressure. In ulcerative lesions of the lower part of the

larynx, especially the vocal cords, the pain is likely to be absent, or moderate in degree. Coughing and talking may then be moderately painful. Rubbing together of ulcerated false cords on coughing and talking produces pain. Ulcers about the arytenoids are painful, especially because of movement. This is true in all the upper part of the larynx. In proportion as the ulcer approaches the upper opening of the organ, and particularly if it involves the epiglottis, the resulting pain, especially from swallowing, is the most severe produced by this disease. Its intensity is often comparable with that caused by the passing of a calculus through the ureter.

The general symptoms of laryngeal tuberculosis are usually obscured by the accompanying symptoms of pulmonary disorder. If the latter disease has not yet actively developed, slight increase of temperature and pulse rate, with a certain degree of general weakness are likely to be present. When the disease is limited to a small area, however, and not acute, no general symptoms are to be expected from the laryngeal disease alone.

Diagnosis—Differential Diagnosis. (153)—The presence of pulmonary tuberculosis, if known, serves to direct the suspicions of the laryngologist towards laryngeal tuberculosis. But when pulmonary tuberculosis is not known to be present, and not at the time discoverable, the diagnostician is, nevertheless, by no means warrranted in excluding from his consideration local tuberculous disease. In either case he will find it necessary from a differential standpoint to consider especially chronic simple laryngitis, syphilis, lupus, and, rarely, carcinoma. In certain cases two, or even three of these diseases, including tuberculosis, may be present in the same larynx. The history often serves to shape the conclusion and general tests, as that with tuberculin, or the Wassermann reaction may be very important. The study of general symptoms, or of local symptoms elsewhere in the body, may be the factor that settles the doubt. Our view of the diseased larynx, as has been said, is always relatively hampered. The time when the diagnosis is of greatest importance is at the inception of the disease when the diagnosis is hardest to make. A study of the disease as to its manner of progress, or as to its response to treatment, may also be necessary before a clear diagnosis is possible to be made.

The distinctive features between Tuberculous Laryngitis, Chronic Simple Laryngitis, Syphilitic Laryngitis, Carcinoma of the Larynx and Lupus of the Larynx may be seen in the following tables:

	A. TUBERCULOUS LARYNGITIS	B. CHRONIC SIMPLE LARYNGITIS (Catarrhal Laryngitis)
Age of Patient	Usually young individuals.	Not important as to age.
General Symptoms	Are usually present; slight, or severe; depend on condition of lungs.	Are usually absent; possibly those of accompanying bronchitis, or pharyngitis.
Onset and Early Manifestations	The mucous membrane apt to be generally pale; lesion small, localized, monolateral; posterior part of larynx; elevation, or ulceration; voice hoarse, weak or normal; slower progress than syphilis.	A generalized hyperemia, is likely to be bilateral. Not ulcerated; hoarseness; moderate swelling.
General Tests	Wassermann is usually negative. Tuberculin may be positive. Tubercle bacillus when present, may have come from pulmonary and not from laryngeal sputum.	Wassermann and Tuberculin tests usually negative; tubercle bacilli absent.
Pain or Ulceration	Is due chiefly to ulceration; depends on location of ulcer; most severe when posterior and high.	Is rare; ulceration is absent.
Lesions	More rapid than carcinoma; less rapid than syphilis; lesions tend to become multiple, with multiple areas of ulceration. Stenosis of larynx rare.	Rarely produces irregular localized infiltration; usually a generalized moderate swelling.
Treatment	The effect of antisyphilitic is negative. Treatment: General systemic and local as given in the text, chiefly paliative to relieve pain.	Difficult of cure. Paliative and local. Not affected by antisyphilitic treatment.

	C. SYPHILITIC LARYNGITIS	D. CARCINOMA OF THE LARYNX
Age of Patient	Not important, but most often young; may be child.	Over thirty.
General Symptoms	Signs of disease elsewhere in body; lymphatic glands enlarged; history of genital infection and progress (if obtainable).	At first absent; later, loss of weight, weakness, and cachexia.
Onset and Early Manifestations	Depends on whether early or late lesion. If tertiary, localized elevation of mucous membrane; may be anterior; often rapid growth and quick ulceration; voice may be hoarse.	Localized elevation of mucous membrane, gradually growing into actual tumor; growth slower than tuberculosis, or syphilis.
Tests	Wassermann test positive; tuberculin test usually negative; tubercle bacilli absent.	Tuberculin and Wassermann negative. Tubercle bacillus absent.
Ulceration	Tertiary ulcer is typically deep, with elevated border, and sharp cut edges; rapid development.	Relatively late in developing; apt to be shallow and without much secretion; characteristic odor.
Pain	Less characteristic than in tuberculosis, but may be severe and constant.	Varies, but may be severe when ulcerated, a relatively late symptom.
Lesions	Gummatous swellings that develop rapidly, and quickly, ulcerate deeply.	Lesion develops usually as a defined tumor mass, infiltrating deeply and eventually closing larynx.
Treatment	Quickly improves under antisyphilitic treatment.	Antisyphilitic treatment produces no effect unless to reduce the pain temporarily. Operative, if life saving. Paliative to relieve pain and suffering.

E. LUPUS OF THE LARYNX

Age of Patient
Young usually; may be a child.

General Symptoms
Practically absent.

Onset and Early Manifestations
Very slow and insidious. Usually free margin of epiglottis is first attacked; mono lateral; extends downward; anemia not characteristic.

Tests
Wassermann usually negative; tuberculin test usually positive; tubercle bacillus absent; Lupus of face may be present.

Pain
Usually absent; if present slight.

Lesions
Smooth, hard, small nodules; these tend to soften and ulcerate; dry ulcer base; leave scar tissue and distortion; slow progress.

Treatment
Antisyphilitic and general anti-tuberculosis treatment is without effect. Local treatment.

Prognosis

Undoubtedly the present widespread interest in the early treatment of pulmonary tuberculosis has not only reduced the number of cases of laryngeal tuberculosis, but has also served to arrest much incipient laryngeal disease. Formerly laryngologists offered little prognostic hope in the disease. In 1919, Sir St. Clair Thompson (58) of London presented to the American Laryngological Association an important personal study of 883 pulmonary patients at a tuberculosis sanatorium. Of these, 687 were pulmonary, non-laryngeal, and 196 also laryngeal. The condition of these patients was investigated after they had been discharged from the sanatorium for from three to seven years. Of the non-laryngeal cases, 39.7 per cent had died at the end of this period, and of the laryngeal cases 68.9 per cent had died. Sir St. Clair writes: "Amongst all the fairly early cases of pulmonary tuberculosis admitted to a sanatorium, the expectation is that 60 per cent will be alive in 3 to 7 years. But of similar sanatorium patients with the larynx diseased only, 30 per cent will be alive at the end of that period." While this report must not be accepted definitely for all cases as to percentages, it undoubtedly shows clearly the serious prognostic gravity of the laryngeal complication of pulmonary tuberculosis; it also shows clearly that the disease is often recoverable. The seriousness of the prognosis in the individual case is dependent upon the progress and activity of the pulmonary disease, as well as of the laryngeal, and upon the promptness and efficiency of the treatment for both.

Treatment: (A) **Prophylactic.**(176) With respect to getting important results in laryngeal tuberculosis serious difficulties present themselves. But difficulties present themselves in pulmonary tuberculosis as well. Until the internist and laryngologist learn to co-operate more fully many more patients will die of laryngeal complication of tuberculosis than need to. The larynx should undergo frequent and systematic inspection in ev-

ery pulmonary patient. The conception that local measures (in combination with general measures) are capable of producing no better laryngeal results than can be secured by general measures alone is definitely and seriously false.

Moreover, the laryngeal point of view is important even to the internist. For, as movement tends to encourage the lodgment of the tubercle bacilli in the larynx, so discouragement of movement tends to prevent such lodgment. This fact places control of coughing, talking, and swallowing definitely in the category of prophylactic measures having reference to laryngeal tuberculosis. Excessive coughing demands sedatives, and such measures of control as deep measured breathing to allay irritation. Coughing also demands study of nasal, post-nasal, and pharyngeal conditions, in order, through local treatment, to eradicate catarrhal, cough-producing inflammations. Talking is subject to the control of the pulmonary patient and should be considerately discouraged by the physician. Even swallowing may be mitigated in a measure as to its movement effects by careful chewing and swallowing of food, and by discouraging eating merely for pleasure and not for nutrition.

Another manner of pharyngeal prophylaxis should be in the mind of the general physician, and should inspire of itself examination of the upper air passages; that is, the possibility that chronic lingual or faucial tonsillitis, rhinitis, nasal sinusitis, pharyngitis, may, by encouragement of simple chronic laryngitis, be also encouraging laryngeal tuberculosis. The neck in pulmonary patients, on similar grounds, should not be coddled, but rationally protected from exposure to the cold; excessive dust in the air should be guarded against. Smoking tends to produce dryness of the laryngeal and pharyngeal mucous membrane, and thus to encourage sticking of laryngeal secretion, with consequent cough, and is, therefore, from a laryngeal point of view, to be completely interdicted in all pulmonary patients, with or without laryngeal tuberculosis.

(B) **General and local measures.** The general measures of treatment of laryngeal tuberculosis are those also of pulmonary tuberculosis. Tuberculin[2] has been much employed, but has

[2] Specific therapy in laryngeal tuberculosis. The use of tuberculin. As pulmonary tuberculous disease generally causes the main disturbance and laryngeal tuberculosis is most frequently only secondary, tuberculin may be most advantageously used if the indications for its use are advised in the treatment of the ordinary pulmonary disorder. Should, however, the treatment with tuberculin be here contraindicated, that is when the laryngeal disease is the more disturbing factor, then it has no place as a therapeutic remedy.—J. R.

failed the medical profession of the essential value in laryngeal tuberculosis.

When one approaches consideration of local treatment he is at once up against another real difficulty. This has two phases. All local laryngeal treatment is difficult and requires much skill. In some patients anatomical conformations, especially with reference to the epiglottis, render both observation and manipulation of instruments unusually difficult; while in certain patients, the extreme sensibility of the throat to the employment of instruments may render local treatment so difficult and so trying to the patient as to practically prevent more than the minimum of such treatment.

Again reverting to prophylaxis, it is to be realized that whatever tends to encourage fluidity of the secretions that must pass outwards through the larynx is desirable. The tuberculous patient whose throat readily admits of the effective employment of instrumentation, the use of simple alkaline sprays to the larynx by the patient himself, is to be encouraged, whether the patient has or has not developed local laryngeal disease. Another measure that should tend to liquify secretion is the employment of oily, non-irritating sprays that stimulate the secretory glands of the mucous membrane and the use of oil inhalations could be readily arranged as a routine treatment for all pulmonary patients. Menthol, oil of cloves, of wintergreen, or phenol, in liquid petrolatum, are examples of suitable drugs for such a purpose.

When tuberculosis has definitely made its appearance in the larynx, and there exists hope of its arrest, talking should cease completely, at least until the disease has come under control. The matter of local infection is extremely serious, and abstinence from talking is one of the most important measures. If recovery is beyond hope, or when recovery has practically been secured, the amount of talking may be regulated in accordance with local sites and manifestations of the disease and other individual considerations. Whispering is much less harmful than loud talking, and may be permitted when loud talking should be forbidden.

Coming to the subject of local medical or surgical treatment, we are confronted with two more or less definite attitudes on the part of the laryngologists. Speaking briefly, one group says, use as little local treatment as you have to, inhibit talking, strengthen

general measures so far as possible, and then when local treatment is definitely demanded, use it with emphasis. The second group says, watch the larynx closely, inhibit talking, employ local antiseptics and caustics intelligently and persistently, as the conditions warrant. There is room for argument on both sides. Persistent use of chemical antiseptics and caustics in incipient, localized cases are often curative, after general measures have proven their ineffectiveness. This is a matter of repeated experience. To argue that the general measures should first be permitted to prove their ineffectiveness before taking up local treatment, is to argue in favor of taking chances, owing to the progress of the local and general disease, that local treatment may be instituted too late. On the other hand, to argue that, whenever local measures are employed, to make them emphatic and at once destructive of the local lesion, may be to argue correctly.

The two classes of treatment most discussed are, on the one hand, antiseptics and caustics with, or without, curettement, and, on the other, the electric cautery, or operation. Undoubtedly both classes of methods are capable, when expertly employed, of producing destruction of the local lesion. The actual cautery, since the application is difficult and must be applied with precision, and is not without danger, demands the greater expertness. Any application to be destructive of disease must be exactly applied. Chemical agents of moderate, but increasing strengths, may, in certain lesions, be equally effective, but require more time. Their advantage is that, either due to the anatomical difficulties presented by the individual patient or to movement, or to relative inexpertness on the part of the operator, no great harm results if the application is caused to extend beyond the area intended to be reached. The antiseptics that may be employed include silver nitrate, argyrol, zinc chlorid, lactic acid, formalin, iodine, and balsam of Peru. Formaline has been found to be especially effective in strengths of from 1 per cent to 10 per cent. Variation of the drug employed in the same patient seems at times to be essential.

The electric cautery is efficient in the destruction of ulcers, infiltrations, and tumor masses. Sprays are of general inhibitive antiseptic value, but are not curative of the tuberculous lesions, as such. Much the same may be said of the laryngeal syringe and of powders. The latter, owing to their effect in increasing the dryness and stickiness of the secretions, should not be used.

A recent author advocates the passage of a rather large cotton swab saturated with a solution of menthol in olive oil between the vocal cords. The resulting irritation to the larynx due to the presence of the foreign body, causes the vocal cords to contract on the swab and squeeze the antisepticized oil out over the larynx. This would also serve for prophylaxis, or inhibitive antisepsis, rather than to destroy existing local tuberculous disease.

Direct and suspension laryngoscopy have afforded the laryngologist a new measure of surgical approach to the larynx that is being employed for larger surgical procedures, as the amputation of tumor masses, the amputation of epiglottis (132), curettement and the electric cautery. Local anaesthesia is usually possible. Careful consideration of the general condition of the patient, in conjunction with the urgency of the operative measures to be employed, are requisite in determining the safety of such measures of treatment.

It is many years since Enos, a general practitioner in Brooklyn, studied a case of laryngeal tuberculosis in which the epiglottis had been entirely destroyed and in whom swallowing had not been interfered with. Lorenzo B. Lockard, of Denver (116), has since devoted much practical study to amputation, in whole or in part, of the epiglottis for tuberculosis. His studies indicate that even infiltrations not uncommonly penetrate into the perichondrium and even into the cartilage. Such lesions are curable only by removal or deep destruction. At first, the operation of epiglottidectomy was performed solely for the pain, and such pain is thereby commonly completely eliminated. More recently the operation has been extended to include relatively incipient epiglottic cases, is undertaken to arrest and cure the local disease, and has won a recognized place in surgery in laryngeal tuberculosis. The operation is performed by direct laryngoscopic methods, either by biting off the organ with forcepts, or, preferably, by removal with the cold wire snare.

For actual laryngeal occlusion, tracheotomy sometimes becomes necessary. But even under relatively favorable general conditions, the operation has had fatal results in a large percentage of cases, and should always be considered as serious. Heliotherapy has proven to be of value (121), but does not in any sense replace other local measures of treatment. The light may be applied from the dermatological surface of the larynx or with

the aid of a throat mirror by actual reflection into the interior of the organ. Higher mountain altitudes are considered to have especial value by observers of experience, but many pulmonary patients find any but low, or moderate altitudes dangerous.

To relieve pain in the larynx, and especially pain on swallowing, is to encourage recovery of both the general and local disease. Of available drugs orthoform, either in powder, or emulsion (Wolff Freudentahl) (53) has produced the best results. Anesthesin and cocaine (in mild spray, or powder) also have their place. Cocaine is too poisonous for extended use. Propaesin is well spoken of. If orthoform is well placed, especially when applied in emulsion by syringe, the effects are marked and prolonged for hours and the drug is not toxic. If needed, it may be repeated before each meal.

The method of nerve blocking of the larynx first advocated by Hoffmann has won a permanent place in the treatment of local laryngeal pain. It consists in the injection of the internal branch of the superior laryngeal nerve with 65 to 80 per cent alcohol. The procedure does not paralyze, and offers no contraindications. The superior laryngeal nerve is the nerve of sensation of the laryngeal mucous membrane, the epiglottis, and a part of the base of the tongue. It is easily approachable. It passes into the larynx through the thyro-hyoid membrane, far to the outside, somewhat under the anterior border of the sternocleido-mastoid muscle. The nerve is not deeply seated, especially in the emaciated tuberculous patient, and may be even less than one-quarter inch from the surface of the skin. The technic of injection described is that followed by Robert McD. Lukens (177). Position of patient: Sitting, head natural position, or supine on a pillow, head to one side. The greater cornu of hyoid and superior cornu of thyroid cartilage are located with the left forefinger. Needle (strong and blunt) enters about one inch anterior to directing finger, and one-quarter inch above that of the thyroid cartilage; it is pushed backward and inward, the point being raised from time to time, to locate its position by the directing finger tip of the left hand. If patient feels pain directed to the ear of the corresponding side he is previously directed to raise a hand. At that moment a drop or two is injected. If cough results the fluid has entered the larynx; the needle is then withdrawn a little and a few more drops injected; if cough does not ensue, ½ to 1 cc is injected, while moving the tip of

the needle around a little, in order to surround the nerve. Other ways of approaching the nerve are also employed.

One, or both, sides may be injected at one sitting. The injection may be repeated at intervals of three to five days. Relief from a single injection varies from a few hours to a month or more. If successful the cessation of pain is completed. Unfortunately, no way exists by which the operator may be certain that the nerve has been exactly entered, and, therefore, a large proportion of failures must be expected. E. L. K.

CHAPTER 29

TUBERCULOUS PERITONITIS

Peritonitis Tuberculosa. Miliary Tuberculous Enteritis. Tuberculosis of the Peritoneum. Tabes Mesenterica. Tuberculosis of the Abdomen, Etc.

Primary and Secondary

General Consideration. Etiology. (47) (a) Primary tuberculosis of the peritoneum in the adult is extremely infrequent; it is, however, more so in child life. It is usually always secondary, although cases have been observed without any evidence of tuberculosis of other organs or tissues, but such cases are doubtful because the real primary focus either near or remote may have been entirely overlooked, and may have been only pinhead in size and have wholly escaped detection.

(b) Secondary tuberculous peritonitis, that is secondary to tuberculosis of other organs of the body, is the general rule. It usually follows in the wake of tuberculosis of the lungs and pleura, intestinal tuberculosis, tuberculosis of the abdominal glands, the retroperitoneal and mesenteric, tuberculosis of the genital organs, in acute miliary tuberculosis and in the late form of chronic pulmonary disease. In the female a close relationship has frequently been observed between tuberculosis of the peritoneum and that of the genital organs. Tuberculous peritonitis may be primary to a genital tuberculosis—the genital tuberculosis being secondary to a peritoneal or extra genital. Tuberculous peritonitis and tuberculosis of the genital organs may exist at the same time—both may be hematogenous from one and the same focus. A tuberculous peritonitis is most likely never secondary to a previous genital tuberculosis, although many good observers maintain that genital tuberculosis frequently leads, in about one-third of the cases, to a diffuse tuberculous peritonitis and that very rarely the reverse is observed, namely, that a tuberculous peritonitis is followed by a genital tuberculosis. Tuberculous peritonitis is generally a disease of

young or middle life—it is very rarely encountered after the fourth decade.

Clinical Forms (89). Tuberculous peritonitis is usually observed in one of two forms, (1) the acute and (2) the chronic, and the acute permits of a further division into an acute miliary and an acute circumscribed. (a) The acute miliary or generalized form, a hematogenous infection which is usually of short duration—generally both surfaces of the peritoneum are involved, many miliary and submiliary gray nodules as a rule are present, extending over both surfaces of the liver and spleen, and owing to the short duration of the disease but little exudate is present, and if so, it is either serofibrinous, bloody or pus. (b) Acute circumscribed tuberculous peritonitis. The disease is limited or localized to areas with little tubercle formation but much fibrinous adhesions.

(2) Chronic diffuse tuberculous peritonitis. A slow progressive form which may manifest itself as either a dry or moist, sicca or humida variety. In the dry variety owing to a fibrinous more or less plastic exudate, the abdominal organs become matted or glued together, adhesion between the various abdominal organs takes place, and the omentum becomes contracted and is rolled up as a palpable mass in the upper abdomen. This is the peritonitis tuberculosa adhesiva of the older writers. The exudative or moist variety, the peritonitis tuberculosa humida, is characterized by the presence in the abdomen of fluid which may be serous or serofibrinous, purulent or bloody. Adhesions between the intestines and the abdominal viscera are usually present. Many tuberculous nodules are found on both walls of the peritoneum. As a rule they seldom calcify or caseate and not infrequently such tuberculous nodules are found on the peritoneum surrounded by a hemorrhagic area, or as happens in older cases, the nodules are encircled by much dark pigmentation derived from the blood. The amount of fluid present may vary from a few cc to that of many litres and it may be either free in the abdominal cavity or found localized, that is, sacculated. If the peritonitis is the result of tuberculous enteritis, acute septic peritonitis results. Usually in the abdominal exudate the tubercle bacillus may be found.

Symptoms of Tuberculous Peritonitis. They are present according to the nature of the inflammatory process. Symptoms of (1) acute, and (2) chronic peritonitis. (a) Acute miliary

form. Owing to the extremely rapid progress of the disease the symptoms are quite variable—all followed by a swift overwhelming of the organism and early exitus. (b) Acute circumbscribed or localized form is usually accompanied by fever and pressure pains, diarrhoea or constipation, friction sounds, marasmus, etc. (2) Chronic form, dry or moist. Symptoms of pain in the abdomen, loss of appetite, a feeling of weakness is present with a fulness in the abdomen, alternating pain from the spine to the abdomen, a bloated feeling, nausea, occasionally vomiting, a body loss and an abdominal gain, particularly noticeable by the patient. Meteorism due to the lessening of the natural parastalsis, eructation of gases, vomiting, etc. If vomiting is accompanied by a fecal odor, much distension of the abdomen, colicky pain and constipation, ileus may be suspected due to intestinal paralysis or obstructions from inflammatory contractions. Much gas or exudate in the abdomen may lead to dyspneic symptoms. Fever may be present or absent, the pulse and respiration both rapid and occasionally there is a more or less pronounced jaundice present and the bowel movements are colorless. This is especially so in children with diarrhoea. The disease may be slow and insidious in its course with constant remissions and exacerbations. The appearance of the abdomen may differ greatly according as the dry or moist variety predominates. If the exudate is absorbed, thickened bands of the peritoneum are palpable and, on the other hand, if there is free fluid in the abdomen by percussion and change of position, it may be outlined and the amount of fluid approximately ascertained and if the amount is large and it is withdrawn or aspirated, the nodular or tumor-like masses in the abdomen become palpable. In cases in which the mesentary is contracted, a high tympanitic note is frequently elicited over the right abdomen, due to gaseous distension of the small intestine, while over the left the note is dull and flat. Death is due usually to asthenia or suffocation. Spontaneous cures have been observed and arrest of the tuberculous process is not infrequent. The duration of the disease is variable. In about 50 per cent the disorder lasts from one to six months, in 25 per cent from six months to one year and in the remaining 25 per cent from one to five years, and not considering complications which may arise, such as ileus or perforation or perhaps the acute miliary form, the disease runs a more or less protracted course. In a child a tuber-

culous peritonitis may also be either acute or chronic. In about one-third of the cases the disease is of the acute form and in the beginning it may be most difficult to differentiate it from appendix disease, typhoid, etc. Ascites in children generally indicates tuberculosis of the peritoneum and here again by exclusion we must differentiate from cirrhosis of the liver, late hereditary syphilis, obstruction of the hepatic vein and chronic inflammation of the peritoneum, pleura and pericardium. Tuberculous peritonitis may be accompanied by pleurisy and in the chronically protracted cases, more or less inflammation of many serous cavities, known as polyseristis, polyseritis, polyorrhomenitis, Morbus Bambergeri, etc., may be present. This is probably due to the disposition, which means a difference in the nutritious soil, a difference in the virulence or perhaps a difference between the disorder produced by the tubercle bacillus and that of the toxins. A gastro-intestinal disturbance followed by frequent and repeated colicky attacks extending over prolonged periods, loss of appetite, constipation and emaciation, distension of the abdomen and meteorism, face pale, slight temperature, abdominal pain, especially if the abdomen becomes tense, point to tuberculous peritonitis.

Diagnosis (Differential). The diagnosis is not always easy. It may present many difficulties and may simulate many non-tuberculous disorders. Peritoneal friction sounds if heard near the region of the heart may be mistaken for pericardial murmurs. Exudative tuberculous peritonitis may be confused with cirrhosis of the liver, thrombus of the portal vein, carcinoma and sarcoma, ovarian cysts, ascites from unknown causes or following chronic heart and kidney disease and with chronic non-tuberculous peritonitis. The chronic course of the disorder, the pain, the changing fever, the exudate, the gradual emaciation and diarrhoea point to tuberculous peritonitis, particularly if taken in connection with tuberculosis of other organs. Three facts, an abdominal tumor, ascites and emaciation with anemia indicate tabes mesenterica. During the menstrual period the fever usually rises. In alcoholic subjects tuberculous peritonitis and cirrhosis of the liver may exist at the same time, because alcoholism favors both. In many instances the diagnosis is dependent upon either an exploratory puncture or laparotomy. In the non-exudative or dry form if an exploratory laparotomy is contraindicated, the diagnostic use of tuberculin may be resorted

to. Usually with the intradermal use of tuberculin in one or two milligram doses in a case of suspected tuberculous peritonitis, increased abdominal pain following the injection strongly points to abdominal tuberculosis. Tuberculous peritonitis may escape notice entirely until complications reveal the true and serious nature of the disorder. In children chronic peritonitis is generally tuberculous, and only occasionally due to injury or to extension from a non-tuberculous focus. A high fever at the onset would suggest a pneumococcal rather than a tuberculous peritonitis—hence a severe generalized peritonitis without any definite cause, great and rapid distension of the abdomen, pain and tenderness, high fever and diarrhoea, is usually pneumococcic, while a round, tense distended and shining abdomen with full veins, fluid in the flanks, resonance or tympany about a protruding umbilicus, pain, tenderness and fever is usually tuberculous.

Prognosis. (20) In pronounced phthisis a tuberculous peritonitis is always unfavorable, exitus letalis in 96 per cent. Cases of peritonitis due to an intestinal ulcerative process with pus and pouring of the contents into the abdominal cavity are usually fatal. The dry form of the disease gives generally a better prognosis. Tuberculous peritonitis complicated by cirrhosis of the liver is early fatal due to the want of resistance from the generalized tuberculous condition. In about 15 per cent of tuberculous peritonitis, cirrhosis of the liver is found as an accompanying disorder, and in other cases of cirrhosis of the liver, frequently towards the terminal stage, a tuberculous peritonitis is noticeable. The longest duration of the disease is five years. Spontaneous cures are rare and the remissions frequent. Alcoholism is a frequent etiological factor with a grave prognosis. Ascites with pronounced tuberculosis elsewhere, constant diarrhoea, extreme emaciation, cutaneous hemorrhages, also presages a grave prognosis. In mixed infection the outlook is always bad. Relapses after latency or an apparent cure are frequent and are less common in children than in women with tuberculous salpingitis. In general the prognosis is better in children than in adults, better if the ascites is serofibrinous in form. If other organs are involved or if the onset of the disease is very acute the prognosis is less favorable; the dry form better and ulceration with pus very grave. Primary intestinal tuberculosis in early child life is less frequent than the path-

ologic respiratory tuberculosis as a primary disorder, but as to prognosis is just as unfavorable.

Therapy—Surgical and Medical

Surgical Treatment. When to treat surgically? In every active, in the so-called hopeless cases. The usual surgical procedure is by laparotomy opening of the abdomen, removal of the fluid and incidentally of the appendix or a diseased ovary. Laparotomy is performed with the purpose of bringing about a reactive hyperemia and to stimulate the bactericidal action of a 10 per cent tincture of iodine freely applied to the abdominal cavity seems to be sufficient. Many surgeons apply iodoform to the peritoneum and the abdominal organs after the fluid has been withdrawn and before the abdomen is closed. The use of a 10 per cent Tincture of Iodine freely applied to the abdominal viscera and about the wound is often followed by most gratifying results, or aspirating, by means of a trocar and canula, the free fluid—this followed by washing of the inner abdomen with a sterile physiological salt solution after which about 100 cc of a 20 per cent iodoform emulsion are injected. In peritonitis tuberculosa sicca the instituting of an artificial ascites by means of injecting about 30 grams of glycerine into the peritoneal cavity has met with most favorable results. The injections must be repeated at long intervals. The use of hydrogen peroxide, a 50 per cent solution, flushing of the abdominal cavity and intestines followed by a physiological salt solution is highly recommended and good results have followed the injection of pure and filtered oxygen into the abdominal cavity in amounts a trifle less than the amount of fluid withdrawn. Laparotomy is always indicated when the exudate is profuse and there is much dyspnea, when the exudate is caseous or purulent, when the lumen of the ileum has become contracted from inflammatory causes or when a tumor of the generative organs accompanies the tuberculous process. It is not advisable to operate too early if a healing of the tuberculous process is desired, corresponding in this particular with pleurisy with effusion of which it is the analogue where if the fluid is removed too early reaccumulation rapidly takes place. Therapeutically laparotomy will always give the best results if applied in conjunction with general treatment.

Medical Treatment. When to treat the peritonitis medically?

In the less acute cases, first and foremost, as in tuberculous diseases of other organs, institute the dietetic-hygienic treatment in combination with a tuberculin therapy. The tuberculin as well as the dietetic-hygienic treatment should be resorted to after an laparotomy. In all cases of tuberculous peritonitis, post-operative or otherwise, the use of old tuberculin in very small doses is always indicated and as a beginning amount give one millimilligram dose and so on every third or fourth day, increasing the dosage gradually until milligram doses are administered or till a marked improvement is noticeable, when a tonic dose may be given once in two or three weeks and maintained almost indefinitely. Warm applications to the abdomen either in the form of hot compresses or warm alcohol on a soft cloth are indicated and often give temporary relief. Remedies which favor resorption of the fluid are always desired and the anointing of the abdomen once a day with mercurial ointment using from ½ to 1 drachm or applying and rubbing gently over the abdomen from ½ to 1 tablespoonful of green soap. As a most satisfactory topical application I have found the use of tincture of iodine painting over the abdomen, repeating it once every three or four days. Camphor in the form of the camphorated oil or spirits of camphor slightly heated and applied is much in use. All digestive disturbances should be met as they arise, constipation by an appropriate anticonstipation diet and enemas, and diarrhoea by a suitable diet and remedies to check the excessive bowel movements. The most distressing symptom usually complained of is the constant feeling of fulness in the bowel, the distension with gases, meteorism. If there is absence of diarrhoea small oft repeated doses of calomel, say 1/10 gr. every hour for ½ dozen doses, are indicated. High colonic flushing with chamomile infusion is a valuable remedy. Salol in 5 to 10 grain doses every three hours, and for pain and distress in the abdomen 15 to 20 drops of Chloranodyne given in a little water or still better in a ½ teacupful of an infusion of anis, fennel or caraway seed. For pain morphine is always indicated either by mouth or preferably hypodermically and alcohol and opium as a topical application. The use of spirits of caraway or the essence of anis or fennel seed may be used. The internal use of the tincture of iodine in milk I have found to be a most dependable drug promising good and lasting results, when given in 20 drop doses three or four times a day. It has been proven that the individual suf-

fering from tuberculous peritonitis is often very greatly benefited by a residence at the seashore, most likely due to the minute quantities of iodine contained in the air. The arsenical preparations and the various iron tonics are always admissible. Drugs like guaiacol and creosote although formerly much in use and which may have proven meritorious in the hands of some are now obsolete.

Tuberculous peritonitis as the immediate cause of death is extremely rare, some intercurrent disorder usually ending the scene. The expectant and the operative treatment show about the same mortality. Usually surgical interference is indicated when the course is rapid and progressive as in asthenia, cardiac weakness and hectic fever, and if the conservative treatment has been without favorable results, otherwise, it should be the rule in all cases of exudative tuberculous peritonitis as long as the symptoms are those of a plain serous fluid even if there be much ascites to treat such cases for long periods of time simply conservatively. It is immaterial whether the tuberculous peritonitis is simply of the serofibrinous variety or whether it is also accompanied by ascites, though perhaps the latter is more quickly and more promptly influenced by the treatment. In the treatment of tuberculous peritonitis, whether surgical or medical, the object is to bring about a process of healing by inducing hyperemia and favoring transudation and adhesions.

CHAPTER 30

TUBERCULOSIS OF BONES AND JOINTS

General Consideration. Tuberculosis of the bones and joints is a metastatic infection from a focus elsewhere in the individual, is always secondary, except for purely suppositious cases in which the track of tuberculosis may be a stab wound or other artificial means. Ordinarily it is an hematogenous infection. Little masses of tuberculous material from caseating glands or from other sources get into the blood current and are carried along until they find a favorable resting place. This place may be in normal tissue, or it may be, and is more likely to be, in tissue which has been bruised or injured just enough to cause a small local hemorrhage or stasis, as in the cancellous portion of the bone. Here the tubercle bacilli become arrested and begin to form typical lesions which can be seen as very small grayish spots in the cancellous bone. As these spots increase in size and number the centers become necrotic and caseous, and the bacilli escape and set up new foci in the surrounding bone. (36) (49).

The most frequent site is in the epiphyses, near the joints but not actually in the joints; and, if treatment be successful, the disease may be arrested or cured without any invasion of the joints themselves. This result, however, is very rare, and in most cases the joint is sooner or later involved, with more or less destruction of the articular surfaces. The diaphysis, or shaft, of the bone is almost never invaded. Between the epiphysis (where the blood stream is more active and where the growing of the bone is largely accomplished) and the diaphysis exists a dense, and strong cartilaginous epiphyseal plate which is not readily penetrated by epiphyseal disease. This forms a barrier to the extension of the disease in that direction, so it is easier for tuberculous material to find its way into the joint. In exceptional instances, however, primary joint tuberculosis has been observed, where the synovial membrane was first infected, but these are exceptions.

The subject of tuberculosis of the joints is of great importance

for several reasons; it is a very common disease; it is a very chronic disease; and it is a very disabling disease. The economic loss, in time and money, is enormous. The pain, suffering and disability to the individual patients are distressing.

Joint tuberculosis is primarily a disease of children. More than 85 per cent of all the cases occur in children from two to

Fig. 51. Tuberculosis of the knee joint. Anterio-posterior view (Girl 8 years old. Duration of disease 2 years) (From The Children's Memorial Hospital—H. J. Ullmann, Roentgenologist)

ten years of age, and more than 50 per cent in children from two to five years. In about 40 per cent of all cases the disease occurs in the spine, in about 30 per cent in the hip, and in 20 per cent in the knee.

Symptomatology and Diagnosis of Bone and Joint Tuberculosis.

The general symptoms of bone and joint tuberculosis are those of a slowly developing inflammation. The incidence is always slow, and this is one of the most important points in differentiation. In very small children the trouble may begin in

the small joints of the hand or foot, but it is usually seen in the spine, knee, or hip. Ordinarily in the larger joints of the body, the first symptom that one can distinguish objectively is limitation of motion. For example, a child has a tuberculous inflammation near the hip joint; the first thing that nature does is to try to limit motion in the joint; consequently as the child walks he cannot move his hip joint as freely as the other one, and the result is that he limps a little. Where tuberculosis of

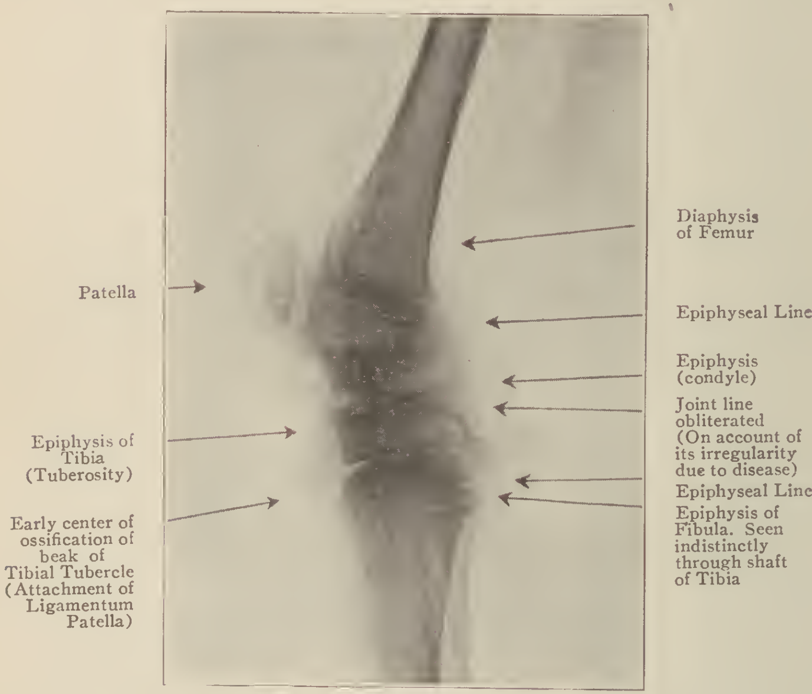

Fig. 52. Knee Joint Tuberculosis. Same case as Fig. 51. Lateral view

the larger joints is accompanied by a limp, this is the first noticeable subjective and objective symptom. A little stiffness comes first, and the first thing the parent notices is this little limp, hyperextension of the hip joint being the first thing that is lost. The first objective sign that a skilled person can notice is a restriction of the mobility of the joint and the first that lay people notice is a limp due to the restriction of mobility. Similarly in cases of tuberculosis of the spine, the first thing that is noticed is that the spine is held stiffly. When the child bends

over to pick up an object from the floor he does not bend his back and curve it forward in a typical case of Pott's disease but he walks up to the object and bends his knees without bending his back. This is due to the fact that nature is trying to hold the spine still. If the child attempts to bend the spine forward it makes pressure upon the bodies of the diseased vertebrae, which causes pain, and it instinctively protects itself against the possibility of pain by holding the back stiff. These symptoms of joint tuberculosis are very often misunderstood, and the diagnosis is often incorrectly made. Persistent limitation is the commonest of all the signs in joint diseases.

Disease of the spine is by far the most important, and we see the largest number of cases of spinal tuberculosis in the clinics. There are a great many more joints in the spine than there are in the knee and the hip. The spine is linked together with the seven cervical, the twelve dorsal and five lumbar articulations as possible sites for the disease. The first thing noticed about the child is that he holds his back stiffly, that he does not bend it freely. It is rare for a child with an oncoming tuberculosis of the spine to complain of any pain in the back. They do not feel pain consciously. They know there is something wrong with the back if they are old enough to reason at all, but they do not feel much actual pain. The mother is very apt to believe that because the child holds his back stiffly there is not anything the matter with it; that there must be some other site in the body for whatever trouble the child may have. The first thing the physician, however, observes is a characteristic rigidity of the spine that makes him suspect tuberculosis. Suppose it is a very early case and the child does not hold his back so very stiffly, but there is some limitation of motion. If the child be old enough and we ask him to bend forward, to bend either side, to bend backward, observing the child while going through these motions, we can usually see there is an area of rigidity in the back. A more accurate way of testing spinal rigidity is to have the child lie face down on a table; then as he lies comfortably face down, pick him up by the two ankles and curve his spine backwards, hyperextend it. With his body lying flat on the table, his hips and legs being curved upwards, naturally the back itself begins to bend. In the case of very early Pott's disease of the spine we notice at once that above and below the region where we think there is disease the spine bends

perfectly freely, but at the site where the trouble exists there will be an area involving three or four or perhaps five vertebrae that does not bend. There is as yet no kyphosis, no knuckle or gibbus in the spine, and all you can find is a little rigid area in an otherwise normal spine.

Other symptoms which come on usually a little later are night cries. Almost all children with joint tuberculosis, whether it be in the spine, hip or knee, will have what are called night cries. The child goes to sleep, and as he is going to sleep, nature is exercising some control over that diseased joint, is causing a reflex spasm that holds the joint more or less stiff. As the child lapses into a deep, profound slumber, the muscles relax a little bit, and the child perhaps rolls or turns in bed, the joint is moved or twisted a little bit and instantly reflex action takes place. The muscles jerk and hold the hip or spine stiff again, and the child is awakened subconsciously with a cry. He gives a loud, shrill cry, but he does not know that he has done so and is asleep again immediately. It is a typical tuberculous night cry. It is a fairly constant symptom in joint tuberculosis.

Symptoms of Reflex Pain. The next symptom observed is a reflex pain. In children, particularly, pain is never complained of in the place where the joint lesion exists. As a rule if a child has tuberculosis of the spine he never complains of pain in his back. This, however, does not hold good in adult tuberculosis of the spine because adults do have pain at the site of disease. This reflex pain is in the direction of the distribution of some of the nerves which supply this region. For example, in a mid-dorsal case of spinal tuberculosis, the intercostal nerves which come down from that region supply the upper part of the belly, and the child has bellyache. He has no disturbance of digestion, his bowels are regular; he has no flatus, no colic, none of the things that ordinarily produce bellyache in children, but he does have pain in his abdomen. It does not come on with any reference to his meals, and the pain is ordinarily thought to be due to indigestion or colic. In a case of stomach-ache in a child do not be contented by simply looking at the child's belly, or perhaps examining the appendix region, but turn him around and look at the spine and you may find a sharp enough kyphosis or knuckle to be readily seen.

If the trouble be higher up in the spine the pain will be noticed there and one may think of heart disease, lung tuber-

culosis or something else as the cause. If they be lower in the lumbar region, pains shooting around, down the front of the pelvis and sometimes down the thigh will be experienced. We see in cases of hip joint tuberculosis that the patient never complains of pain in the hip but always in the knee, and on the inner and front side of the knee, never on the outer.

In spinal tuberculosis, the next sign that soon follows is a prominence of the spinous processes of the vertebrae at the site of the disease. The reason that these spinous processes become prominent is because the bodies of the vertebrae are eaten away, the vertebral bodies collapse and the spinous processes in the back form a sharp bend.

When the disease has eaten away a portion of one or two of these vertebral bodies and it is usually a single one that is attacked first, the spine begins to tip forward and a sharp little knuckle appears in the back. It is always a sharp knuckle, never a rounded kyphosis and this is one of the points in differential diagnosis.

There are many cases of curvature of the spine or scoliosis in which there is a backward as well as a sidewise curvature, and we must ascertain if it is tuberculosis or not. A pretty clear history from the parents or friends that originally the tuberculous knuckle started as a little, sharp protrusion is characteristic. If a sharp kyphosis develops in a child's back without a history of fracture of the spine, which is the only other thing that is at all common in children, you can be reasonably sure that you have tuberculosis of the spine. In addition you will find that the muscles up and down the back within four or five inches of this area are stiff, spastic; that is, they have a reflex spasm which is holding the spine stiff; this is very constant, and this little spasm of muscles sometimes precedes any of the other symptoms. Therefore, a stiffness of the spine in a localized area, with a little projection, in the absence of the history of sufficient traumatism to cause a fracture is diagnostic of Pott's disease.

Differential Diagnosis. There are other conditions that may produce kyphosis in a child's back. Scoliosis does not produce a sharp, but a rounded knuckle which may simulate an old Pott's disease, because the longer the case progresses, the more vertebrae are attacked, and the more rounded the kyphosis gets.

Fracture of the spine is the only thing that we practically ever see. A definite syphilis of the spine, although extremely rare, a gumma, may cause a condition macroscopically very much like a Pott's disease. Here other signs of syphilis, and a Wassermann or spinal fluid Wassermann may help in the diagnosis. New growths, sarcomas, etc., may start with a fairly sharp knuckle, but almost always involving at least two vertebrae in the beginning. Hypernephroma has at times caused a necrosis of the spine, but the hyperhephromas, by the time they make a kyphosis, have such a large tumor in the belly that one does not think of Pott's disease in that connection. Those are the only other things that make a very sharp irregularity in the child's back.

As a broad general rule we may say that if a child has a sharp knuckle in the back, with a little stiffness of the spine, and perhaps a little referred pain, it is almost sure to be a case of Pott's disease.

Complications. The chief complication to be dreaded in tuberculous joint disease is the formation of tuberculous abscesses commonly called "cold" abscesses. These can sometimes be prevented by early and careful treatment, but occasionally occur in spite of every effort. They are perfectly harmless unless they become so large as to interfere with the wearing of proper apparatus or unless they become contaminated with other kinds of bacteria. The reason they are so dangerous is because physicians are apt to open and drain them under the mistaken impression that the contents of the abscess may be doing harm to the patient. In some instances the abscesses open themselves. Now, when a cold abscess is opened, it almost immediately becomes infected with the ordinary pus-producing micrococci, usually the staphylococci, in spite of the most careful methods of dressing, and in a few days this infection travels up along the walls of the cavity and into every nook and cranny. This, in the case of the large psoas abscesses, means that a very considerable area within the body is now infected with active pyogenic cocci instead of being merely a reservoir for broken-down tuberculous material. The patient quickly responds to this infection, and develops fever, exhaustion and loss of weight, in addition to more rapid destruction of the joint and the neighboring tissues. It is, therefore, of

great importance to prevent the opening of such an abscess whenever possible. It may sometimes be done by repeated aspiration of the contents, through a needle or small trocar, with perhaps the injection of iodine or a mixture of camphor and phenol. The method advised by Cheyne, open incision, evacuation, and suture in layers, is usually followed by failure, since the abscess, after refilling, almost always opens spontaneously through the site of the incision.

Another complication of tuberculous joint disease is the development of tuberculous meningitis, which is invariably fatal. It is, of course, possible that the meningitis may develop from the original focus of infection, but there is some evidence against this theory.

A complication often seen in spinal tuberculosis is paralysis, due to the pressure upon the spinal cord of a cold abscess or of the products of inflammation of the membranes which cover the cord. The paralysis is almost always spastic in type, and occurs, usually, only in the dorsal and cervical cases. It comes on gradually, with weakness of the legs and increasing difficulty in walking until finally the patient becomes entirely unable to walk. It can be cured in all but a few cases by prolonged rest in bed, with fixation of the spine by apparatus or operation.

Treatment of Bone and Joint Tuberculosis. (Conservative and Operative.) (A) Conservative Treatment. The local treatment is of the greatest importance. It takes at least two years for a tuberculous hip or knee-joint to get well, and at least five years for a tuberculosis of the spine, under conservative treatment, and it may take two or three times as long when the patient's resistance is poor. The time may be shortened materially by operation in those cases which are suitable for operation.

It is a well-established fact that tuberculous joints will get well if they are kept still for a long enough time. This means that some form of apparatus must be applied which will hold the joint in a fixed position so that it cannot move. The more perfectly it can be immobilized, the sooner it will get well.

(1) **The Treatment of Spinal Tuberculosis.** This differs somewhat from the treatment of the other tuberculous joints. In children if we hold the spine still long enough the tuberculous process will disappear and this is true in practically every case.

In acute and painful disease of the spine, the best method for rest is by absolute recumbency in bed, upon a "Bradford frame" made of gas pipe and covered with tightly stretched cloth. The frame is a few inches longer than the child, and two inches narrower than the width of the shoulders, and webbing straps are passed diagonally over the shoulder on one side and under the axilla on the other side, and buckled beneath the frame, so as to hold the thorax firmly to the frame. A folded towel is passed around the pelvis and pinned under the frame. This holds the spine almost immovable, and, in the case of Pott's disease, requires thick pads on either side of the kyphosis (or hump) to prevent the skin from becoming excoriated by pressure. The routine that is necessary in practically all these cases of Pott's disease is to lay the child on a frame and fasten it down so that it cannot get away. Every morning the child is carefully rolled over and the spine washed off with alcohol and ordinary talcum powder put on the back. This is a daily routine so long as they are in recumbency. Sometimes they are kept on such a frame for two or three or more years or until they become perfectly well.

The further treatment is very simple, only to keep the spine still long enough. Ordinarily it takes four to five years for a tuberculous focus in the spine to get well. If after four or five years the child has no muscle spasm in the back, there is no sign of a tuberculous abscess, there is no paralysis, there is no referred pain, then the child may be well. We then place the child in an apparatus, in a brace or a cast. If the disease be above the mid-dorsal region this will not be sufficient unless it includes the head. The head, chin, occiput have to be supported if the disease be above the sixth or seventh dorsal vertebra. We let the child up in this brace, increasing the time it can be upright until it is upright practically all day, and then we begin to take off the brace for a little while every day, and if then the child has no return of symptoms for six months or a year we consider it is well and we then let the child go without any support. Suppose, on the other hand that we have treated the child as long as it can be treated in bed; is it right to get it up in an apparatus any sooner? It probably in a good many cases can be gotten up sooner than two or three or four years if the mechanical principles can be carried out; if we can

make forward pressure on the knuckle and have enough leverage above to push back on the child's chest and pelvis, we can accomplish a good deal. It becomes then simply a plain question of mechanics. If the disease is high up, there is not a brace that has ever been made that will prevent a deformity. Tell the parents that no brace will prevent the deformity from increasing; that no cast will do so; that if the child is going to be upright it will have more deformity, also tell the parents perfectly frankly the exact proposition; that if they do not want the child to have a hump it must stay in bed two or three years on a frame. On the other hand, if they do not mind a hump, the child may be up and about in a brace.

The general condition also has to be attended to. The child should live an outdoor life, and if possible, be rolled over so that the sun's rays will shine on the back every day. (See Chapter XXI, B.—Heliotherapy.)

(2) **Hip and Knee Joint Tuberculosis.** Cases of hip or knee joint disease are treated in the same way during the acute stage, with the addition of Buck's extension by weight and pulley to correct whatever flexion deformity may be present. After a few months of bed treatment, it is advisable in most cases to get the patient up in some form of immobilization apparatus, because the difficulty and expense of prolonged bed-treatment are almost prohibitive in the majority of cases. Many ingenious and efficient braces have been designed, the most useful being the Taylor brace for spinal disease, the modified long Thomas-Phelps traction splint for the hip cases and the Thomas knee-splint for the tuberculous knees. In some clinics, plaster of Paris is used for almost all cases.

Most American surgeons believe that tuberculous hip joints and knee joints should be protected from weight-bearing, to prevent destruction of the joint-surfaces, but there is considerable evidence that such joints are not apt to be very useful functionally, and that perhaps a speedy ankylosis is, after all, the quickest and best result to be obtained. The general rule should be that in children every effort should be made to obtain a useful, movable joint in hip and knee. If the disease goes on to suppuration, and becomes secondarily infected with pus germs, it may be necessary to produce a rapid ankylosis in order to avoid the consequences of prolonged septic exhaustion.

(B) **Operative Treatment.** There is a procedure that will hasten recovery, and that is an operative stiffening of the spine, making the spine its own brace. In 1912 Albee and Hibbs (178), wrote about their experience of ankylosing the spine. Albee's (36) idea was to split the spinous processes with a chisel, turning half to the side so as to make a V-shaped groove along the diseased area of the spine, two or three vertebra above and two or three below. Into that cleft which was made, a narrow strip of bone taken from the tibia with a chisel or a motor saw was sewed in place with kangaroo tendon or chromic catgut, keeping the child recumbent until body ankylosis took place. This is an extremely useful operation but it should not be done in the presence of sepsis; it should not be done if there is a discharging sinus, because after the operation infection may occur and the bone graft will be lost. I am prepared at the present time to recommend this operation for any case over seven or eight years of age, with the understanding that children will have to be treated by a brace or by recumbency for at least two years afterwards, because the ankylosis which forms does not consist of very rigid bone, and in young children the bone may bend so that the deformity will increase unless carefully protected. We must not allow parents to think that the Albee operation will cure the case radically without any further treatment. Until children are twelve or fourteen years of age the bones have to be supported or the deformity will get worse.

The Hibbs operation consists in paring off the periosteum from the spinous processes and the laminae, and the articular facets and laying strips of bone which are cut from the laminae themselves alongside of the spine, and nipping off the spinous processes with a bone cutting forceps so that each spinous process will lie down on the one below it, making a very complete and solid ankylosis of the spine by this method of fusion. In very young children who need the support of a bony ankylosis a Hibbs operation is advisable in preference to the Albee method.

Tuberculosis of the Spine in Adults. In adults who have had for a long time a pain in the back at a definite site, always suspect something organic. Do not think it is a neurosis or hysteria. Many cases are treated for neuroses, hysteria and men-

tal conditions who have perfectly typical Pott's disease. Remember, adults do not have pain about the belly, girdle pains, to the same extent that children have; they have a pain at the site of the trouble, that it is a constant, severe pain; that they do not get kyphosis early because the vertebrae are hard and solid; children get kyphosis early; adults late. The X-ray plate is not helpful in bone and joint tuberculosis until actual bony, destruction has taken place, although it sometimes shows fairly definite bone atrophy. Adults may go for years without a perceptible knuckle or before perceptible X-ray findings are discovered, although a definite tuberculous spondylitis may have existed during all of this time. The first thing known about an adult spinal tuberculosis is sometimes a rapidly oncoming paralysis, a spastic paraplegia.

Adults may also show an early abscess, a psoas abscess, which is the last thing that the average practitioner thinks about in a case of back pain. If a man has had pain in his back for a good while and you think he may have tuberculosis of the spine, do not neglect to feel of his abdomen. If a patient has any kind of symptoms, do not hesitate to have him remove his clothes. Many mistakes are made in perfectly simple cases by modesty on the part of the doctors or the patients themselves in removing the clothes for the purpose of examination.

In adults, tuberculosis of the spine demands a different kind of treatment from that in children and tuberculosis of the hips and of the knees also demands a different treatment. By children we mean cases under twelve or fourteen years of age, by adults everything beyond that. Children will get well with protection of the joints for a sufficient length of time, but adults with joint tuberculosis, whether it is in the hip, spine or knee, will not get well by either protection or conservative treatment. A man with Pott's disease or with knee or hip joint tuberculosis will not get well by holding him in a brace even if for many years. Joint tuberculosis in adults does not tend to get well without operation.

In adults with Pott's disease we ankylose the spine by the method of Albee as soon as we can. In adults with hip joint

tuberculosis we also operate as soon as we can. Here we do what is known as an arthrodesis which means a stiffening of the joint. For example, we find tuberculosis in the hip joint; we cut down on the joint, resect the trochanter temporarily because that is the easiest way of getting into the joint and with a chisel we scoop out the acetabulum; with the same chisel square off the head of the femur; put these two together and place the patient in a plaster cast. In ten or twelve weeks he will have a good ankylosis of his hip joint. In this way he will get well with a stiff hip, able to do his work, in nine or twelve months; whereas if we did not fasten up the hip joint, at the end of five, ten or fifteen years he would be still under treatment. In the same way we excise the knee joint for tuberculosis and cure in a few months cases that have been treated with casts and braces for five or ten years. I have seen many people with knee-joint tuberculosis who have been treated conservatively for from ten to seventeen years by competent orthopedic surgeons, and who were no more nearly well after this prolonged treatment than when the treatment was begun. I have never seen an adult with a proven tuberculous knee get well without operation. Where we do a rapid, conservative arthrodesis, excision of the joint, taking away only a thin strip from the head of the tibia and the femur, putting the bones together and applying a cast, in a few months the individual gets a good stiff knee joint. There is a difference between the forms of treatment in children and in adults. As a rule be as conservative as you can, and as long as you can in the treatment of children; in the adults be as radical as you can, as soon as you can. We see people going about year after year with painful swollen tuberculous joints and spines, and comparatively soon after operations we see them well, the joints solid, free from any manifestations of tuberculosis and the patients able to go about and earn their living. Remember to suspect tuberculous joint disease in children whenever they have limitation of joint motion without any apparent sign, and also remember further that the way to get well is to make the joint stiff as soon as possible whether this be done by artificial apparatus or by operation.

Tuberculin. The question of tuberculin treatment is one of great importance. An experience extending over many years

has verified the facts that cases which get minute doses of tuberculin do better than those which do not, and so it should be a routine in the treatment of bone and joint disease. Tuberculin should be administered hypodermatically (to small children it may be given by inunction according to the method of Petruschky) beginning with most minute doses according to the direction given in Chapter XXIV, "Tuberculin—its therapeutic indications."—E. W. R.

CHAPTER 31

TUBERCULOSIS OF THE GENITO-URINARY ORGANS

General Consideration. Tuberculosis involving the genito-urinary tract in its early stages begins in either the urinary organs or the genital organs. Later in the course of the disease, or sometimes when the patient presents himself for examination, one finds that both of these tracts are involved, hence the use of the phrase—genito-urinary tuberculosis. In the cases in which the tuberculous lesion is located in only one or the other tract, it would be better to speak of it as genital tuberculosis or urinary tuberculosis. With this object in view the subject will be considered under these two headings.

(A) Tuberculosis of the Urinary Tract

Tuberculosis of the urinary tract, according to present day conception, always begins in the kidney, that is, the tuberculous process is usually primary in the kidney as regards the urinary tract. The exception to this are the cases in which late in the course of the disease the tuberculosis reaches the other kidney via the ureter.

(1) Tuberculosis of the Kidney

Frequency. Renal tuberculosis was formerly considered a rare lesion of the kidney. With the improvements in modern diagnostic methods, the cases are more and more accurately diagnosed, so that there is an apparent increase in this disease. Kapsammer, (185) who examined and reported 20,770 autopsies, was able to find renal tuberculosis present in about 1 per cent. Wildbolz (96) states that in 2,345 autopsies reported from the Pathological Institute at Berne, renal tuberculosis was found in 5.3 per cent, and in this same series evidence of pulmonary tuberculosis was found in 20.7 per cent.

Age. Renal tuberculosis is essentially a disease of adult life, the largest number of cases occurring between the ages of 20 and 40. Cases occurring in children and young adults are not uncommon, and doubtless many escape recognition.

Sex. Renal tuberculosis occurs with about equal frequency in both sexes. Statistics vary on this point, however, and these

differences may be due to the diversity of the material of the various investigators. More women are operated upon than men but more men come to autopsy than women, so the disparity may be due in part to these facts.

Side Involved. Statistics show that the right kidney is more often the seat of tuberculosis than is the left, which is probably due to the increased mobility of the right kidney. In Küster's (148) statistics there were 189 cases of right-sided tuberculosis as compared with 163 cases of left-sided.

Predisposing Factors—(a) Traumatism. It is questionable whether traumatism in the strict sense of the word is a predisposing factor in the production of renal tuberculosis. Because of its depth it is difficult to see how a patient could sustain a trauma that would predispose to the localization of tubercle bacilli without producing gross kidney disturbance.

(b) Stone. The influence of stone in the production of renal tuberculosis does not seem to be very great. Primary stone and tuberculosis is a rare combination, but secondary stone is occasionally found in a tuberculous kidney. That the relationship existing between the two cannot be very great is evidenced by the fact that tuberculosis rarely develops in a patient who has previously been operated upon for renal stone.

(c) Pyelitis. It is believed that chronic pyelitis may be a predisposing factor, but this is probably not true, as many patients who are diagnosed as cases of pyelitis are in reality cases of renal tuberculosis which are not recognized at the time.

Pathogenesis. The present view regarding the origin of urinary tuberculosis is that the condition is primary in the kidney and that the process extends from the kidney down the ureter to the bladder, so that bladder tuberculosis, therefore, is secondary to kidney tuberculosis. Kidney tuberculosis is, of course, secondary to a primary focus elsewhere in the body, such as lymph glands, lungs, bones, joints, tendons, or synovial membrane.

The theories regarding the origin of renal tuberculosis are generally considered under (a) hematogenous, (b) lymphogenous, and (c) urogenous.

(a) Hematogenous. The present day views are almost unanimous in holding that renal tuberculosis is a blood-borne disease, that is, that the tubercle bacilli are carried to and deposited in the kidney by the blood stream.

(b) Lymphogenous. Various lymphatic theories have been advanced. (See Chapter III.)

(c) Urogenous. That tubercle bacilli may be promulgated up the ureter by the urinary current is possible. Cases of secondary tuberculosis of the bladder with ulceration, great frequency of urination, associated with straining and increase in intravesical pressure, are frequent. It has been shown that bladder urine backs up into the ureter, so that it is easy to see that urine from the bladder could travel up the ureter of the healthy kidney and carry with it tubercle bacilli, if the ureteral orifice is ulcerated or rigid and gaping.

Symptoms—(a) **Kidney. Tenderness to Pressure.** This may be variable and may be demonstrated by ordinary palpation or by deep pressure with the thumb placed in the angle made by the last rib and the deep lumbar muscles.

Great care, however, must be used in evaluating this symptom, as there is the possibility of this tenderness being manifested on the side of the healthy kidney, when it may be due to an enlargement or compensatory hypertrophy of the normal kidney.

(b) **Colic.** Renal colic associated with or as a symptom of renal tuberculosis is sometimes due to the presence of calculi. In the largest percentage of cases there are really incrustations of necrotic tissue in the tuberculous cavities. In other instances, one may find incrustations of tuberculous ulcerations in the pelvis.

(c) **Bladder Symptoms—Frequency of Urination.** Frequency of urination may be the first symptom observed and one of its characteristics is its nocturnal occurrence, which may necessitate arising once or twice each night, or the patient may be disturbed as often as every fifteen to thirty minutes. Frequency is sometimes associated with great urgency, so that the patient is obliged to respond at once or the act becomes involuntary. As a rule, the onset of this symptom is gradual and progressive.

(d) **Pain.** As a result of the tuberculous involvement of the bladder, the bladder mucosa becomes sensitive so that the act of micturition is painful. The pain usually radiates along the urethra, but may be localized in the bladder.

(e) **Pyuria.** Pus in the urine is a more or less constant symptom. The amount of pus may vary from one examination to the other, so that a specimen examined one day may show a great deal, and a second specimen examined the next day may be relatively free from pus.

(f) **Hematuria.** This is not always present, especially early in the disease, although tuberculosis is one of the most frequent causes of hematuria. The blood may be microscopic in amount, or it may be so great as to overshadow and obscure all the other symptoms of renal tuberculosis. The hematuria may be so profuse and continuous that the life of the patient is endangered, and knowledge of the fact that the hemorrhage was due to tuberculosis is first obtained when the kidney is removed. Termi-

Fig. 53. Tuberculosis of the Kidney Showing Early Lesion in One Papilla—(a) with Extension of the Process to the Mucosa of the Pelvis

nal hematuria with the passage of small blood-stained shreds of pus is seen more frequently than is profuse hematuria, and it occurs generally at the end of urination and is associated with a great deal of pain.

Pathology. Renal tuberculosis occurs in one of two forms, either as an acute miliary tuberculosis or as the so-called chronic renal tuberculosis. The acute miliary tuberculosis is a part of a generalized miliary tuberculosis in which the process is demon-

strated throughout the entire body, and as such possesses no surgical possibilities and does not fall within the scope of this chapter.

Chronic renal tuberculosis may be more or less arbitrarily classified as follows:

1. The large cheesy, cavernous type.
2. Tuberculous ulceration of one or more renal papillae.
3. Chronic, discrete nodular type.
4. Fibroid type.

In addition, there has been described a so-called miliary tuberculosis of the kidney proper, a so-called acute miliary renal tuberculosis, but it does not represent, as a rule, the early stage of a so-called chronic renal tuberculosis.

Not infrequently more than one of these forms may be seen in the same kidney. In other words, these various types are not always as sharply defined as may appear from this classification

Much speculation has been put forth regarding the part of the kidney in which the tuberculous process begins. Among the early cases operated upon, the most advanced lesion found has been in one of the renal papillae, either alone or with an involvement of adjacent calices. It was formerly believed that papillary tuberculosis was a special form of renal tuberculosis, but since cases began to come to early operation, those in which the papillae seem to be the first site of involvement have been noticed more and more. This, of course, is the result of early diagnosis. It is believed by many that all cases of renal tuberculosis begin as tuberculosis of the papillæ.

In the early cases, the kidney appears to be normal upon inspection. The cut surface shows changes in one or more papillae and the papillae corresponding to them may show the presence of tubercles, and at times, small areas of caseation may be seen. In the more advanced cases, the surface of the kidney shows the presence of tubercles, which may be arranged in groups. There may be more than one group and, as a rule, normal appearing kidney tissue can be found between the groups of tubercles, which often project above the surface of the kidney and are visible to the naked eye. The organ may or may not be slightly enlarged. Tubercles that may have the appearance of infarcts and areas of caseation may be seen on cut section. Between the areas of caseation may be seen large cavities with

thick walls which are often more or less irregular in outline. The walls are covered with thick, caseating masses, and the cavities are sometimes lined with thick, mucous granulation tissue. In the caseous tissue surrounding the cavities, tubercles may be found. Most of these cavities communicate with the renal pelvis.

As the cavities increase in size, they enlarge toward the cortex of the kidney, the cortex becomes extremely thin and the

Fig. 54. Tuberculosis of the Kidney Advanced. (Phthisis of the Kidney.) Extensive Involvement of Both Upper and Lower Poles, Showing Large Thick Walled Cavities—a, in Lower Pole

cavities protrude above the surface of the kidney, so that the kidney surface becomes irregular, the kidney increases in size and loses its characteristic shape. Because of the loss of kidney tissue, it assumes a pale color. At this stage, tubercles are infrequently seen on the surface, being limited to the parts of the parenchyma that have not as yet undergone extensive change.

Further increase in size is dependent in part on the condition

of the ureter. If the kidney becomes strictured so as to interfere with drainage of the urine and pus, the kidney may reach an enormous size, resulting in the production of a so-called tuberculous hydronephrosis. If the ureter remains patent, so the drainage is not interfered with, the kidney may undergo loss of parenchyma due to destruction by the tuberculous process, and it becomes very small, at times weighing not over 30 to 60 grams.

The coverings of the kidney are sooner or later involved in the pathologic process. The fibrous capsule becomes thickened and adherent to the cortex as well as to the fatty capsule, and the changes in the coverings of the kidney may not advance beyond this point. At times, however, the kidney is surrounded by thick, dense, fibrous tissue of almost cartilaginous hardness, due to the fusion of the fibrous and fatty capsules. This process of sclerosis often takes place at the expense of the fatty capsule and the fat remaining is often very fibrous. This thick, fibrous perinephritis is not typical of tuberculosis, as it is also seen in other chronic renal infections.

Suppuration outside of the kidney may occur with the production of a tuberculous perinephritic abscess. This, however, is rare. The renal pelvis, as a rule, is involved early in the course of renal tuberculosis, and the earliest lesions are found in the region of its papillae. Tubercles are seen beneath the mucous membrane and tend to remain localized; later the process spreads over the entire pelvic mucosa. The tubercles undergo caseous degeneration with resulting ulcer formation and the pelvis undergoes dilation due to obstruction in the ureter, which rarely escapes infection, and, as a rule, is involved early. Miliary tubercles may be seen in the mucosa, arranged in small groups between which normal mucosa is seen. These ultimately undergo caseation and form ulcers. The walls of the ureter show thickening and the ureter becomes firm and rigid. There are times when the kidney has a normal appearance. Stricture formation in the ureter is not uncommon. In rare instances, the entire lumen is dilated, resulting in the production of a closed tuberculosis pyonephrosis, which has been termed **auto-nephrectomy.** In such cases the products of infection do not reach the bladder and the patient passes clear urine.

The bladder may remain free from involvement for a long time and the earliest bladder changes are those seen around the ureter of the affected side. The ureter shows hyperemia and is

surrounded by tubercles which are undergoing caseation and ulceration. The process extends from the ureter, and, in cases seen early, tuberculous ulcerations are seen in the corresponding half of the bladder. Later, the entire bladder shows the presence of tuberculous ulceration. Normal mucosa may be seen between these areas and the process is not limited to the mucosa, but involves the submucosa and muscularis, as a result of which the bladder undergoes secondary contraction and has limited capacity. The ureter on the corresponding side often is retracted and becomes dilated. This condition has been termed **"golf hole ureter."**

General Symptoms. Early in the course of the disease there may be no symptoms. As the disease progresses, general symptomatic manifestations present themselves. The patient complains of loss of appetite, he is below par, his strength is not up to the average, and a certain amount of anemia develops. A slight rise in temperature may be noted, which later becomes almost constant, doubtless due to the presence of a mixed infection. It is only late in the course of the disease that marked, general symptomatic manifestations are present. The diagnosis of tuberculosis is occasionally disputed because of the general well-being and general appearance of the patient. The general condition of the patient is not always an index of the amount or extent of the tuberculous process in the kidney, because patients with well-developed or advanced renal tuberculosis often have no evidence as regards their general condition.

Diagnosis. (a) **General.** Perhaps a large number of cases of renal tuberculosis are not recognized, or the possibility of renal tuberculosis is not being thought of. A persistent pyuria should always arouse suspicion of a possible tuberculosis, which may often be strengthened by the presence of tuberculosis in other parts of the body. Scars in the neck indicate tuberculosis of the lymph nodes. A history of long suppuration involving the bones, evidence of joint and tendon tuberculosis, and the presence of tuberculosis of the lung are facts that should not be overlooked. In males, the evidence of a nodule in the prostate, the testicle, or epididymis is very valuable confirmatory evidence. In women, involvement of the tubes and ovaries is rare, whereas in men involvement of the genitals is common.

Palpation may not give any additional information except for tenderness over the bladder, and if bladder tenderness is asso-

ciated with a history of painful distention and exquisite sensitiveness to instrumentation, much valuable information is obtained.

(b) **Urinalysis.** Albuminuria is a constant finding. It has been said that renal tuberculosis does not exist without the finding of albumin in the urine and that its absence almost excludes the possibility.

A persistent pyuria that does not yield to the usual treatment within a reasonable time should always call for special methods of examination in order to exclude the possibility of tuberculosis. The pyuria is often small in amount and not much significance may be attached to its presence and hence early recognition of the cases fails. Large amounts of pus are always found late in the course of the disease. The urine may be free from pus in cases of so-called closed pyonephrosis.

(c) **Tubercle Bacilli in Urine.** While many of the symptoms enumerated above may suggest the possibility of renal tuberculosis and a presumptive diagnosis be made, the final diagnosis can only be made upon the demonstration of tubercle bacilli in the urine[1]. It was formerly taught that tubercle bacilli were difficult to demonstrate in the urine, but this old dogma has been disproven. (See Chapter XXXVIII—"The Urine in Tuberculosis.") It has been variously estimated that in cases of renal tuberculosis, the bacilli can be demonstrated in upwards of 90 per cent of the cases in direct smear. Undoubtedly, tubercle bacilli can be found much more readily in cases in which the bladder has become involved, that is, late in the course of the disease.

Great care must be exercised in examining for the Koch's bacillus, to exclude contamination by the smegma bacillus. This is of prime importance. A large percentage of errors can be eliminated if one thoroughly prepares the patient, and then carefully obtains a catheterized specimen from the bladder, or better still examines a specimen obtained by ureteral catheterization in which event the danger of contamination by smegma bacillus hardly enters into consideration. See page 510.

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[1]The demonstration of the Koch's bacillus in the urine is not indisputable evidence of genito-urinary tuberculosis. A bacillemia due to the tubercle bacillus may result in kidney permeability—this without any kidney lesions, simply the effect of the produced toxins circulating in the blood serum when the bacilli may pass through the kidney structures. Permeability to albumin in the supposedly normal kidney is shown in those cases designated by clinicians as pretuberculous albuminuria (see chapter XI) in which the albumin content of the urine increases with the increase of the tuberculous activity in some near or remote part of the organism and tubercle bacilli themselves may pass through a weakened and irritable kidney tissue incident to the toxins and contaminate the urine.—J. R.

(d) **Animal Inoculation.** Guinea pig inoculation with urine of a patient suspected of having urinary tuberculosis is of value in confirming the clinical diagnosis. The objection to this method, however, is the fact that it takes so long before the tuberculosis develops in the guinea pig.

Various methods of guinea pig inoculation have been recommended. The intraperitoneal injections have the advantage in that the pigs develop tuberculosis early, but have the disadvantage that very frequently the pigs die because of a mixed infection, and when this occurs in cases in which the urine is obtained by ureteral catheterization, it means another ureteral catheterization.

Intrahepatic and intrasplenic injections have been advised. In order to shorten the period of incubation in the pig, the preliminary treatment of the pig by x-ray has been suggested. Crushing the inguinal glands and injecting the sediment directly into them has become the method of choice. Ebright has suggested sensitizing the pigs by the previous administration of large doses of tuberculin.

(e) **Diagnostic Injections of Tuberculin.** The injection of old tuberculin for diagnostic purposes may not offer much in the way of an aid in the diagnosis of tuberculosis in the genito-urinary tract. It has been claimed that after a preliminary injection of tuberculin, tubercle bacilli can be found in the urine much more easily and much more frequently, and often in cases in which persistent search has failed to find them. Focal reactions at the site of the disease are supposed to occur after injections of tuberculin. It is questionable, however, whether one would be willing to remove a kidney because of the production of pain in the region of the kidney following tuberculin injections.

Special Methods of Diagnosis. (1) Cystoscopy and Ureteral Catheterization. Cystoscopic examination is the one single method that gives us the most important information, though in early cases it may be negative. In cases in which tuberculosis of the kidney is suspected and in which the patient has been repeatedly cystoscoped with negative findings, one may finally be rewarded by the finding of changes around one ureteral orifice. The early changes are hyperemia and tubercles, and later areas of ulceration.

Ureteral catheterization enables us to determine the origin of the disease, whether right or left side, as well as to establish the

origin of the tubercle bacilli. Furthermore, by means of this method the presence of a second kidney and whether or not it is involved in the tuberculous process may be determined.

Since these group of cases have been more intensively studied by this method of examination, the percentage of cases showing bilateral tuberculosis has increased. It was formerly taught that all cases of renal tuberculosis were unilateral early in the course of the disease. Recent statistics, however, show that from 10 to 15 per cent of the cases of so-called early tuberculosis of the kidney are bilateral.

Cystoscopic examination in early cases is simple and easy. Later, it is difficult because of the exquisitely painful condition of the bladder, when an anesthetic must be resorted to.

In advanced cases, the entire bladder mucosa is red, there are many areas of ulceration, shreds of mucous are seen adherent to the bladder wall and the ureteral orifice is dilated. When the areas of involvement are small, normal mucosa can be seen between the groups of tubercles and ulcerations.

(2) X-Rays. Routine examination of all patients suspected of having renal tuberculosis has shown that many of these patients show definite roentgen ray evidence. Areas of calcification are seen in the kidney region, and in cases of so-called "putty kidneys," the entire kidney outline can be seen. While the x-ray gives corroborative evidence, it should not supplant cystoscopy and ureteral catheterization, because the presence of calcification on one side does not necessarily mean that the tuberculosis may not be bilateral and that there may not be an involvement of the other side.

(3) Functional Kidney Tests. Various functional tests have been advocated from time to time. The phenolsulphonaphthalein test is perhaps the one most relied upon today. A study of the function of the kidney which is to remain is important from the standpoint of first, demonstrating the presence of the kidney; second, of attempting to establish the functional capacity of this kidney—whether or not it will be able to carry on the work of the two kidneys, and third, whether or not infection is present.

(4) Chromoscopy. Injection of indigo-carmin, first advocated by Felker and Joseph, is used by some to determine the functional capacity of the two kidneys, and in this way it obviates, in a certain number of instances, the necessity of ureteral catheterization. After a solution of indigo-carmin has been injected

intramuscularly, it is eliminated by the kidneys. The diseased kidney eliminates the dye slower than does the well side, because it does not possess the power to eliminate as much dye as the well kidney, hence the urine from this kidney is pale blue, whereas the urine from the well side is dark blue. (See Chapter XXI.—A—Chemotherapy.) This test is of value not only in the study of the functions but as an aid in finding the ureters.

Differential Diagnosis. Differential diagnosis may be considered under two headings (a) differentiation from other lesions of the kidney and (b) differentiation from other lesions of the abdomen.

(1) Lesions of the kidney other than tuberculosis. The lesions of the kidney that most frequently come under consideration are (a) stone, (b) tumor, and (c) pyelitis. In the largest number of cases, (a) **stone** can be excluded with good roentgen ray technic and with careful reading of the plates. It must not be forgotten, however, that there are a certain number of stones that cannot be demonstrated by means of roentgen ray. At times, areas of calcification in a tuberculous kidney call for differentiation from stone, and it must not be forgotten that in rare instances stone and tuberculosis may occur in the same kidney. While the clinical symptoms of (b) **tumor** and renal tuberculosis are not very often confused, a differentiation may occasionally be called for, which can be done by means of pyelograms. The routine use of pyelograms in renal tuberculosis, however, is not a desirable procedure and should not be used. (c) **pyelitis.** Many cases of pyelitis are in reality cases of renal tuberculosis. If pelvic lavage is instituted in the treatment of pyelitis and the desired result is not obtained within a reasonable time, careful search for tubercle bacilli and guinea pig inoculations should be made.

(2) Lesions of Other Abdominal Viscera—(1) Gall Stones. Patients with right renal tuberculosis are often mistaken for cases of gall-stone disease. The history of severe abdominal colic, radiating to the shoulder blade, and the presence of dyspeptic symptoms, coupled with the absence of urinary symptoms should lead us to suspect gall-bladder disease rather than renal tuberculosis, in which case bladder symptoms, albumin and pus in the urine are such constant findings.

(2) Appendicitis. The most frequent mistake made is diagnosing cases of renal tuberculosis as cases of chronic appendi-

citis, as a result of which a perfectly harmless appendix is removed and the patient fails to obtain relief from his symptoms. In cases of chronic appendicitis, one should be able to elicit a history of repeated attacks of acute appendicitis—a history which is classic. Sudden onset of pain, reflex nausea and vomiting, and temperature is a symptom-complex that is not common in renal tuberculosis. The presence of pus or bacteria in the urine and the long duration of the patient's symptoms argue rather for renal tuberculosis and against the so-called chronic appendicitis.

(3) Pelvic Disease in Women. Next to appendicitis, the most frequent source of error is made in favor of pelvic lesions in women. Right sided tubal disease and right sided disease of the ovary are the conditions most frequently diagnosed, and because of the simplicity of vaginal examination and the finding of slight disease of these organs the patients are erroneously operated upon. When the pelvic findings are small or insignificant and there is pus in the urine and a history of long-continued bladder distress, these patients should be subjected to careful, painstaking urologic examination before they are operated upon for tubal or ovarian disease.

The Course of Renal Tuberculosis. Renal tuberculosis generally pursues a very chronic course. Not infrequently patients give a history of onset dating back eight or ten years. On the other hand, the course is rather rapid in some cases, in certain ones of which the tuberculous process extends rapidly through the entire genito-urinary tract.

The duration of life after the diagnosis has been made has been variously estimated. There are cases known to be alive ten or fifteen years after the diagnosis of renal tuberculosis was established. Kornfeld (188) found 15 out of 200 cases that lived longer than six years.

Prognosis. In renal tuberculosis as regards the infection in one kidney the prognosis is good. It must, however, be guarded because of the possibility of the tuberculous lesions being present in other parts of the urinary tract. Many women who have been operated upon have subsequently become pregnant and the one kidney may carry the patients through pregnancy without any difficulty.

Causes of Death. The more frequent causes of death are renal insufficiency and cachexia associated with myocarditis and myeloid degeneration. Tuberculosis outside of the genito-urinary

tract is often the primary cause of the deposit of these tubercles. A patient with tuberculosis of the genito-urinary tract is apt to be particularly susceptible to tuberculous meningitis.

It has been agreed that spontaneous cure does not take place, though cases of auto-nephrectomy, as mentioned above, are sometimes cited as examples of spontaneous cure.

Treatment. The treatment is prophylactic, hygienic, and dietetic. The same rules regarding the management of tuberculosis in other parts of the body apply to the management of cases of bladder and kidney tuberculosis. Included in the prophylactic treatment is the avoidance of infection in the urinary organs which might prepare the soil for invasion by tubercle bacilli. In this category should be placed the treatment of urethral strictures, so as to insure free drainage, and the prevention of gonococcus infections.

Tuberculin. There are no proven cases of cured tuberculosis of the kidney *per se* that have been treated by tuberculin, but as an adjunct to the surgical treatment it is a most desirable remedy, hence it should always be used after surgical treatment.

Surgical Treatment. Once the diagnosis of renal tuberculosis has been made the question of surgical removal of the kidney is under discussion, and nephrectomy is the only operation that should ever be contemplated. Unless there is distinct contraindication, such as an active and well-advanced tuberculosis of the bones or lungs, the presence of a severe nephritis in the opposite kidney, a tuberculous kidney should be removed as soon as the diagnosis is definitely established. Among the common causes of death following nephrectomy for renal tuberculosis one may mention hemorrhage, pneumonia, myocarditis and meningitis.

Results of Operation. In early cases, the results of the operation is a very rapid amelioration of symptoms. The results to be obtained are in direct ratio to the duration of the condition, that is, if the patient has had bladder symptoms for two years, it will take about two years after operation for these symptoms to disappear. In other words, cases that are recognized and operated upon early offer the best possibilities for complete disappearance of the bladder condition after the operation. Only too often the patient comes in very late in the course of the disease with the bladder in a bad state of contracture, so that the amount of relief obtained is small. Here, as in any other

surgical condition, the key to the entire situation as regards ultimate cure is early diagnosis.

Following operation, the amounts of urine voided rapidly diminish, so that the output returns to normal. Pyuria disappears in a large number of these cases, and the urine ultimately becomes free of pus. Tubercle bacilli may persist in the urine for a long time after operation, as was proved by Israel (117).

After-Treatment. The after-treatment for bladder tuberculosis depends in a measure upon the amount of bladder involvement. In the largest number of instances, I have not resorted to the use of local treatment, which in so many instances is extremely painful. The instillation of bichloride of mercury, in increasing amounts of from 5 to 20 cc and increasing in strength from 1:10,000 to 1:3000, has been used but it results in the production of very much pain. Other local applications that have been recommended are 5 per cent guaiacol or guaiamar injections into the bladder; a warm solution of 6 per cent carbolic acid has also been advised, but has occasionally been followed by hematuria. In order to hasten the cure of tuberculous ulcerations, bladder fulguration and radium have been advised. A secondary operation on the bladder may be necessary in cases in which the symptoms persist in spite of the removal of the kidney. In women, a vesico-vaginal fistula can be carried out; in men suprapubic drainage may be indicated.

(B) Genital Tuberculosis

Tuberculosis of the genital tract is very often confused with tuberculosis of the urinary tract. Many times both occur in the same patient. In cases of tuberculosis of the genital tract there is present, in a large number of cases, definite evidence of tuberculosis in other parts of the body. Thus Barney (169), who has made a careful study of this question, found tuberculosis of other organs in 55.8 per cent of his cases of tuberculosis of the epididymis. The lung was most frequently diseased, with a total of 35 cases or 22.7 per cent. Keyes found a previous extensive tuberculous process in 36 out of his 100 cases. This evidence was most frequent in the lungs, bones, joints, tendons and glands.

Pathogenesis. There is still some discussion regarding the beginning of genital tuberculosis in the male. The largest number of contributors doubtless believe that tuberculosis of the genital tract is primary in the epididymis and that the process spreads from the epididymis to the prostate and seminal vesicles,

and in a definite number of instances, involves the testicle and bladder. The theory that tubercle bacilli are carried to the epididymis by the blood stream seems to be the view accepted by most authors. The fact that other blood-borne infections of the epididymis are seen, such as colon bacillus infections, would seem to lend support to the theory that it is a blood-borne disease. Very often accessory factors have aided in the localization of the process to the epididymis, such as trauma or recent gonorrheal infection of the epididymis.

Whether or not tuberculosis of the prostate is primary, hematogenous, lymphogenous or perhaps deferential, in origin is not a definitely settled question. That tuberculosis in the genital tract begins primarily in the epididymis is not accepted by all, as there are adherents of the theory that tuberculosis begins primarily in the prostate and that it spreads from the prostate to the other parts of the genital tract. Equally unsettled is the question of how the tuberculous process spreads after it has reached the epididymis. Many believe that it spreads by way of the vas deferens. The opponents of this theory point to the fact that the vas deferens may be normal in instances in which tuberculosis is found in the epididymis, prostate gland, and seminal vesicles. Barney (169) believes that the spread is lymphogenous.

Pathology. The tuberculous process begins in the tail of the epididymis. From there it spreads and involves the body and later the head of the epididymis. The process may remain more or less localized for some time, and, indeed, the patient may state that a nodule has been present for many years. Not infrequently the nodule seems to be in a state of apparent healing. Later in the course of the disease, the process spreads to and involves the body of the testicle, the small tubercles increase in size and undergo caseation, and the disease extends beyond the confines of the epididymis and involves the vesicles, which become adherent to the epididymis. An abscess is produced which sooner or later opens externally through the skin and results in sinus formation. The sinus may heal, break open several times or several sinuses may be present. The tunica vaginalis usually presents a tuberculous hydrocele, the vas deferens becomes enlarged and hard, and in the late stages, irregular and beaded; the entire cord may be thickened, and the prostate becomes involved sooner or later. The surface of the prostate is irregular and its consistency

is much increased. The seminal vesicles, in advanced cases, show thickening of the walls, are hard and nodular, and often there may be more or less evidence of a periseminal vesiculitis. Caseous nodules are formed; the wall may undergo fibrous induration so that the vesicle shrinks and produces a hard rigid mass. It is stated that calcification in tuberculous vesicles occurs; this, however, is rare.

Tuberculosis lesions occur with about equal frequency on either the right or left side, and it is believed that they begin on the side corresponding to the involved epididymis. When the case is seen clinically, as a rule both sides are involved.

Tuberculosis of the urethra is rare and occurs late in the course of the disease. Stricture formation occurs rarely, and results in symptoms of urinary difficulty, frequency, and obstruction. In late cases, the bladder is involved. The changes are in no wise different from those described in urinary tuberculosis.

Symptoms. The onset is generally insidious so that the patient cannot state definitely when the disease began, except in the rare instances in which there is a definite and clear cut history of traumatism. An acute tuberculous epididymitis with sudden onset is rare. Although the disease generally begins as a unilateral one, the opposite side tends to become involved sooner or later, and a large number of patients return with involvement of the opposite side after the epididymis first involved has been removed by operation. The fact that the opposite side tends to become involved so frequently is one of the strong arguments in favor of the operation of epididymectomy in which attempt should be made to conserve the testicle.

(1) Swelling. Often the first sign that attracts the patient's attention is a slow, gradual, painless enlargement of one-half of the scrotum which is often associated with increased hardness. The increase in size is occasionally first noticed after traumatism and likewise after an attack of gonorrheal epididymitis. The patient may state that the testicle has never returned to its normal size.

(2) Pain. Pain in the affected side may be absent and is more apt to be present during acute exacerbations. When the testicle becomes very large, there is apt to be not only pain in the testicle but a dragging along the cord and a feeling of weight.

(3) Suppuration. The tuberculous process tends to break down with resulting suppuration and the formation of one or

more fistulae. These may or may not persist, and often the fistulae heal up only to break down, or new fistulae may form in the vicinity. Keyes has stated that suppuration occurs early and rarely appears late.

(4) Urinary Symptoms. Urinary symptoms may be absent for many years. Often it is the symptom that first attracts the patient's attention to his illness. In the early cases, it is doubtless due to the involvement of the prostate and vesicles which accounts for the dysuria and tenesmus.

(5) Sexual Symptoms. Early there may be no disturbance in sexual function, and, even late, the sexual act can be performed and there is no marked diminution in sexual desire. On the other hand, azoöspermia was found by Barney in 85 per cent of the cases. These were cases in which only one epididymis was involved clinically. This doubtless accounts for the high rate of sterility in this group of cases.

(6) Involvement of the Vas Deferens. The vas deferens is involved in the late cases. It is very much enlarged, thickened, hard, beaded or nodular, and the entire cord may show thickening.

Other evidences of tuberculosis are often present, such as scars in the neck the result of an old gland tuberculosis, the presence of active or healed tuberculosis in lung, bone, joints, tendons, etc.

Diagnosis. The history of a painless enlargement of the scrotum and an enlarged nodular epididymis with a beaded condition of the vas deferens, the history of repeated attacks of suppuration, the presence of scars in the scrotum or the presence of a fistula make the diagnosis simple.

Differential Diagnosis. The differential diagnosis calls for differentiation between syphilis and tumors, both of which begin in the body of the testicle. The Wassermann test is of value, in determining the presence or absence of syphilis, although it must not be forgotten that a patient may have both syphilis and tuberculosis of the testicle. Other pus infections of the epididymis should be ruled out, such as gonococcus infections and infections by the colon bacillus, staphylococcus, etc.

Prognosis. The prognosis should be guarded in this as in urinary tuberculosis. According to statistics, 41 per cent of 58 patients died of tuberculosis within six years. The more frequent causes of death are miliary and acute pulmonary tuberculosis.

Treatment. Dietetic-hygienic treatment, including rest, ease of mind, wholesome fresh air, etc., as is so successfully prescribed in other forms of tuberculosis, should be carried out.

Tuberculin Treatment. Much discussion prevails regarding the value of tuberculin in the treatment of tuberculosis of the epididymis. There are many strong advocates for tuberculin treatment who report a large series of apparently cured cases, and there is an equally large group of adherents to the surgical form of treatment, however, tuberculin must be considered a most valuable therapeutic remedy in all post-operative cases.

Surgical Treatment. Three types of surgical treatment may be considered: First: **Incision and Drainage.** In cases in which abscess formation has occurred, simple incision, curetting the cavity and packing it with iodoform gauze is the simplest form of treatment that has been recommended. It is opposed chiefly by the adherents of the tuberculin treatment who advise this form of treatment only when pus is present.

Second: **Conservative Operations.** Epididymectomy is the choice of the operation in that it removes the tuberculous focus and allows the testicle to remain. It is the choice for several reasons, first, because the internal secretion of the testicle is conserved; second, because a large number of patients show involvement of the opposite side. If there is gross evidence of tuberculosis of the testicle, the testicle should also be removed. It has been stated that the testicle can overcome a tuberculous invasion if small in amount.

The operation can usually be performed under local anesthesia, and the epididymis and vas deferens removed **in toto.** Following epididymectomy and vasectomy, it is supposed that the tuberculous process of the prostate and vesicle becomes quiescent and rapidly shows marked improvement. It is, ordinarily, a simple operation, and when carefully performed under local anesthesia, it does not mitigate very much against the patient.

Third: **Radical Operation.** The operation of epididymectomy and vasectomy is opposed on the grounds that it does not remove all the tuberculous infection in the genital tract. Opponents of this operation have suggested a radical procedure, which consists not only in the removal of the epididymis and vasa deferentiae, but, at the same time, removes both seminal vesicles and prostate through the perineum. This rather radical procedure has not met with general favor in this country.—H. L. K.

CHAPTER 32

TUBERCULOSIS OF THE SKIN

General Consideration (24) (25) (26). The subject of tuberculosis of the skin is one of comparatively recent date. Looking back a little over thirty years we find that only one form of tuberculosis of the skin was recognized, but since that time the subject has assumed enormous proportions and from year to year has increased in importance. This undoubtedly is due to the fact that finer methods of diagnosis have been introduced. By the injection of tuberculin intradermally the injection of tuberculous tissue into guinea pigs, the v. Pirquet, the Moro and particularly the Mantoux tests, it has been found that many diseases of the skin are most intimately related to tuberculosis in which this previously had not been suspected. The manisfestations of this disease are as protean as is Syphilis.

There are now four recognized types or varieties of cutaneous tuberculosis in which the tubercle bacillus is usually present in the individual lesion: namely Lupus Vulgaris, Tuberculosis Verrucosa Cutis, Scrofuloderma and Tuberculosis Cutis Orificialis; in addition there are a number of different cutaneous lesions known as Tuberculides, which are ascribed to the toxins produced by the tubercle bacillus generally at some distant focus in the body or as a result of embolisms of dead or attenuated bacilli.

Etiology. The active cause of cutaneous tuberculosis is as elsewhere the Koch's bacillus. Among the predisposing and contributing causes are dust, filth, dampness, overcrowding, poor housing conditions, and unhygienic surroundings, etc. The cachectic, run down and undernourished fall prey to the disease more readily than do the vigorous, healthy, and well nourished.

Types and Varieties. The most important of the tuberculous disorders of the skin is Lupus Vulgaris, the result usually of direct inoculation, although the infection may also be carried to the skin by the lymph currents. The disease most commonly begins in childhood and usually attacks the face, especially the nose and area about the mouth. Children crawling about the

floors of the homes of diseased tuberculous individuals may scratch their faces or pick their noses and inoculate themselves in this way. It has been known to follow vaccination, circumcision, tattooing, piercing the ears for the wearing of ear rings, etc.

A second variety occurring as warty lesions is classified as Tuberculosis Verrucosa Cutis and practically always follows

Fig. 55. Lupus Vulgaris
(Courtesy of Dr. J. F. Waugh)

inoculation. Two forms of this are recognized, one known as the anatomical tubercle or postmortem wart, (Tuberculosis Cutis Necrogenica). This is occasionally observed on the hands of physicians and anatomists. It is intimately associated with the handling of dead human bodies and diseased cattle. It occurs usually on the fingers, especially on the dorsum of the thumb and index finger, as an indurated, horny, pigmented lesion the size of a split pea or a small bean. As a rule it persists as such a lesion for months or years.

The other type is more extensive, more papillomatous, and occurs not only on the hands but also on other parts of the body. These forms of cutaneous tuberculosis are found frequently in certain mining districts where miners, the victims of pulmonary tuberculosis, wipe their mouths with the back of their hands.

Another, the third variety of cutaneous lesion is known as Scrofuloderma. This lesion is an involvement of the skin by direct contiguity from an underlying tuberculous structure such as tuberculous glands of the neck, joints and bones.

A fourth recognized variety is Tuberculosis Cutis Orificialis or Tuberculous Ulcers. These ulcers occur in patients suffering from tuberculosis of the throat and lungs and are generally seen in the mouth, throat, nose and about the anus in patients with gastro-intestinal tuberculosis.

Pathology. Lupus Vulgaris (29) and the other manifestations of cutaneous tuberculosis differ but little in their pathological structure. Their histological appearance is practically identical with tuberculous lesions in other parts of the body. In Lupus the tubercle is found in the deepest part of the corium. In the meshes of a reticulum are seen giant cells, which may or may not contain tubercle bacilli. Surrounding this is a zone of small round cells, with vesicular nuclei, and surrounding these in turn a zone of plasma and young connective tissue cells. The tubercle is surrounded by a capsule of collagenous tissue. The cells lying nearest the center degenerate, their protoplasm becomes homogeneous and their nuclei lose their ability to take a stain satisfactorily. Necrosis and caseation occur as a result of this. The cells at the periphery, however, have a marked power of regeneration and finally become converted into connective tissue. This appears clinically as scar tissue. This explains the occurrence of patches of tuberculous tissue where we see at the same time areas undergoing necrosis and at the same time at their borders areas of scar tissue. In Scrofuloderma the lesion differs in no essential way from tuberculosis of the internal organs. There are present round cells, epitheloid cells and giant cells deep down in the subcutaneous tissue. The nodule degenerates and breaks down and may break through the thin overlying epidermis, giving rise to characteristic ulcers. The degeneration is more marked than in lupus and the bacilli while not numerous are more easily found than in Lupus.

Bowen (165) states that in Tuberculosis Verrucosa Cutis the nodule is situated in the upper part of the papillary layer, often in the papillae themselves.

The epidermis is always secondarily involved. Acanthosis or proliferation of the stratum mucosum is always marked and there is generally an edema of this layer also present. Oftentimes as a

Fig. 56. Lupus Vulgaris

result of the ulceration beneath, as in Scrofuloderma the epidermis is ulcerated and destroyed.

The glands of the skin all suffer in the destructive processes going on in the corium and all may be destroyed.

Diagnosis and Description of the Lesions (122)

(1) **Lupus Vulgaris** is the most frequent as well as the most important of the tuberculous lesions. In about 80 per cent of cases it occurs on the face: in some cases on the upper extremity and in a lesser number on the lower. In 80 per cent of cases it occurs before the twentieth year, and in over 50 per cent in the

first decade of life. Girls are more susceptible than boys, probably because they are more closely confined and frequent the house more.

In the United States or perhaps better in America, Lupus is not at all a common disease: (it is becoming more so particularly in the seaboard cities) practically all of our cases come from abroad. It is common on the continent, especially in Southern Europe and is in a much more virulent condition there. It is also

Fig. 57. Tuberculosis Verrucosa Cutis (Duration 2 Years). This Patient Has Also Laryngeal and Pulmonary Tuberculosis. The Cutaneous Disease began at the Ali of the Nose (Left Side). Author's Patient

common in India and the Scandinavian countries. The parts commonly attacked, in the order of their frequency, are the face, the nose, the cheeks, auricles of the ears, and lips. The disease as it occurs on the face may originate in the mucous membranes and thence extend to the skin, or originate in the skin and later affect the mucous surfaces.

It generally begins as a small nodule, deep down in the corium, only slightly elevated above the level of the skin. In color it

varies from a dark yellow to a yellowish red; it feels soft, is smooth or covered with a fine scale, and occasions no subjective sensations. When pressed with a diascope or glass slide a typical apple jelly brown color is seen. New nodules gradually form and by coalescence of these a plaquė results. The nodules spread by peripheral extension. Their growth is very slow.

In the ulcerative type which fortunately is rarely seen in this country, there is rapid extension with the formation of oval or circular ulcers, having irregular margins and dirty red granulating floors. One type is known where fibrous tissue formation is

Fig. 58. Tuberculosis Verrucosa Cutis

the predominating feature. Often beyond the scars in many cases fresh nodules of the disease are seen.

The scars of Lupus are irregular, indurated, deforming, yellowish white or reddish yellow in color. Many hypertrophic varieties are known. The disease lasts usually for years and relapses are common after what appears to be a cure. The general health may or may not be affected. Sometimes there is a coexistent bronchitis, or laryngeal or pulmonary tuberculosis.

Differentiation. Lupus Vulgaris must be differentiated from the nodular syphilide of tertiary Syphilis, from Blastomycosis, Lupus Erythematosus, and Sarcoid of Boeck.

(a) The Nodular Syphilide occur as multiple, flat, round, circumscribed, firm, dark red nodules, varying in size from a coffee bean to a large nut. Their surface is smooth or desquamating, they are asymmetrically placed, assuming semicircular, reniform or circular configurations and are commonly seen on the forehead, the neck and the buttocks. They begin late in life and the Wassermann reaction is invariably positive.

(b) Blastomycosis also occurs later in life. It begins as a verrucous papule or papulo-pustule that spreads more rapidly than Lupus. It forms distinctly elevated verrucous plaques, dark red in color, with sloping borders which are beset with many small abscesses. In these abscesses the causative organism is found. The lesion is often covered with a heavy crust. Removal of this crust shows a moist verrucous surface covered with papilliform elevations. The lesions of Blastomycosis are commonly seen on the forehead and face, the region about the eyes being often affected.

(c) Lupus Erythematosus, a tuberculide, may at times be confused with Lupus Vulgaris, but its more superficial character, its typical butterfly appearance, affecting the bridge of the nose, and spreading over the cheeks in the form of two wings, affecting the lobes of the ears and the scalp, its greater degree of color, its greasy adherent scales and the dilated follicles with comedone like plugs in them, makes its differentiation easy.

(d) Sarcoid of Boeck also affects the face. It is classed as a tuberculide because of its appearance and histological structure. The lesions may be small or large nodules or plaques. They start as red, edematous or infiltrated spots, which are distinctly elevated, as much as a quarter of an inch, and which burn or itch. They grow purple and telangiectatic centrally, the margin turns brown or yellow, and scales and, as involution sets in they fade, leaving a brownish discoloration and a slight atrophy. They never break down.

(2) Tuberculosis Verrucosa Cutis is a warty condition of the skin, the result of direct inoculation with the tubercle bacillus, and may affect any age. Infection may originate in several different ways. It has followed tattooing, the wound of a sabre in duelling, the handling at autopsy of tuberculous cadavers, and the wiping of the mouth with the back of the hands. The hands, especially the fingers and dorsal surfaces are the commonest sites of infection. The disease begins upon a previous abrasion

as a papule, pustule or vesicle with an infiltrated reddish areola. Plaques, oval or round in shape are formed. These vary in size from that of a small coin to an area several inches in breadth. The patches are verrucous, dry and covered with minute vegetations and crusts. In color they are usually brown, dark brown or purple red and are devoid of sensation. The disease may last for months or years. Complications are rare. Healing is followed by a smooth scar.

This condition resembles in some ways a patch of Blastomycosis. Blastomycosis, however, is generally more moist, the edge is more definitely defined and in the minute abscesses of its border the fungus can be isolated.

(3) **Scrofuloderma** is that type of tuberculosis which affects the corium in the shape of tuberculous abscesses, usually located over a broken down tuberculous gland, bone or joint. It is the result of extension of an underlying tuberculous focus to the skin.

This type is seen most commonly in those who are in poor general health and are the subjects of tuberculosis of glands, bones and joints. It is most common in children and young adults. The lesions are single or multiple; the neck, groins and limbs are the parts commonly affected. The primary lesion is a swelling deep in the corium. As this increases in size the overlying skin becomes reddened, thinned, and eventually breaks down, forming an ulcer. This ulcer has an irregular, oval or linear outline, with a thin, undermined edge. The floor is covered with pale, flabby granulations. The discharge from the ulcer is bloody, purulent or serous and often contains tubercle bacilli. The scars when the lesion heals, are irregular and fibrous. Scrofuloderma is a chronic process and may last for months or years.

(4) **Tuberculosis Cutis Orificialis**, or tuberculous ulcers, are oval or circular indolent ulcers which occur in patients who are the victims of internal tuberculosis. These ulcers occur in the mouth, on the lips, about the anus, and occasionally in the ears. The ulcers are shallow, irregular in shape and size, of a reddish yellow color, and are at times exceedingly painful.

Acute tuberculosis of the skin has been seen in children as nodules, vesicles, bullae, papules and pustules, which form crusted, deep circinate lesions, in practically all of which the tubercle bacillus has been found.

The Tuberculides. Under this title has been grouped those cutaneous eruptions which may occur in individuals believed to be infected with the tubercle bacillus. However, in only a few cases has the bacillus been demonstrated. They react to the various tuberculin tests when applied, and it is believed that the reactions are due to toxins which are derived from some distant tuberculous focus and which are circulating in the blood stream. Another theory recently advanced is that they are the result of embolisms of dead or weakened bacilli, weakened if alive in the struggle they had undergone against the defensive forces of the body in a glandular or joint focus. In many of these tuberculides the histological picture is that of tuberculosis. In this group are included Lichen Scrofulosorum, Folliclis, Acnitis, Acne Scrofulosorum, Acne Cachecticorum,[1] Lupus Erythematosus, Erythema Induratum[2] of Bazin and Sarcoid of Boeck. To this list Highman (14) in his recent work on Dermatology has added another class, tuberculides of presumptive tuberculous nature. In this class he includes Pernios, Pityriasis Rubra of Hebra, Angiokeratoma of Mibelli and a vague group resembling Malcolm Morris' follicular eczema.

(1) Lichen Scrofulosorum occurs in children or young adults. The eruption is a small, round, papular one. The papules are yellowish or brown red in color; in size from a pin head to that of a millet seed. Their summit is often covered with a tiny adherent scale, pustule or horny plug, which conceals a minute depression. The lesions are generally aggregated in oval groups, often have a crescentic form, occur on the trunk and abdomen, and cause no subjective sensations. Their course is chronic;

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[1] A tuberculid occasionally observed in patients suffering from active pulmonary tuberculosis is Acne Variolaformis (89), so called, because these lesions resemble those of variola or small pox. They occur usually in the form of postules tending to necrosis and are upon the forehead, scalp, face, etc. These lesions are seen on young patients suffering from chronic, protracted, but active pulmonary disorder, most particularly in the last months of life.—J. R.

[2] **Erythema Nodosum and Tuberculosis. Etiology and Semeiology.** This form of skin (89) lesion usually accompanies clinically a pulmonary, gland or joint disturbance, a typical exanthematous disease related to joint rheumatism, is supposed to be of toxic origin but is not a so called localized skin symptom of a constitutional or bacterial disorder particularly tuberculosis. It has not been definitely established if there is an etiological connection between erythema nodosum and tuberculosis nor has it been proven that erythema nodosum is the direct result of either the tubercle bacillus or its toxins. It must however be admitted that the lesion is frequently found in children suffering from tuberculosis in one form or another. It is particularly a frequent accompaniment of tuberculous meningitis. It cannot be considered an early symptom nor a grave sign if found in active tuberculous disease. Erythema Nodosum if present may produce a favorable soil which may then activate a latent tuberculous focus, as is observed in measles and other infectious diseases. True Erythema nodosum is found in children exclusively of tuberculous individuals, but the proof that these lesions are themselves of a tuberculous nature is wanting. Erytheme Polymorphe of French writers and other obscure skin lesions usually belong to this same category.—J. R.

they often persist for months or years, and when they clear up they often leave scars.

(2) **Folliclis,** a papulo-necrotic lesion, occurs especially on the hands, feet, elbows, and knees, sometimes on the palms and soles. The primary lesion is a papule or nodule, round in contour, varying in size from a pinhead to a small pea, red or purplish red in color, firm and painless. It is generally surmounted by a small vesicle or vesico-pustule, which crusts over, and when the crust is removed leaves a small ulcer. The ulcer when healed is replaced by a scar. Individual lesions may last only a short time, but crops continue to come and go, and in that way the lesions may last for years.

(3) **Acnitis** differs from Folliclis in that the lesions affect the face, occurring in groups upon the cheeks, upper lip, chin, and forehead. They are red or brown red in color, in size from a pinhead to a pea, firm to the touch, and often capped as are the lesions of Folliclis with a small vesicle or pustule, which upon its disappearance leaves a depressed scar. They also occur in crops.

(4) **Acne Scrofulosorum** consists of an eruption of small red papules, millet seed in size. The eruption occurs principally on the extensor surfaces of the limbs, and the papules as in the other tuberculides, are surmounted by a small pustule which becomes crusted over, forms an ulcer and leaves a scar.

(5) **Acne Cachecticorum** occurs on the face, back, chest, and lower limbs. The lesions are small, pinhead sized papules and papulo-pustules, of a red color. They closely resemble a syphilide. They often persist for years and disappear leaving small, depressed scars.

(6) **Erythema Induration** first described by Bazin as "Erytheme des Scrophuleux" most commonly affects young girls. This eruption occurs on the calves of the legs as deep seated nodules, which gradually break down, forming indolent, irregular shaped ulcers. These ulcers are deep and of variable size, with irregular edges, and a greyish red base. They run a chronic course, resolve spontaneously, and tend to recur.

(7) **Sarcoid of Boeck** occurs as small nodules or as large nodules or plaques. The eruption occurs primarily on the face, shoulders, and upper extremities. The lesions start as red, edematous or infiltrated spots which turn purplish or brownish. They are distinctly elevated, and are the seat of marked itching

and burning sensations. They fade very gradually leaving a brownish discoloration and a slight amount of atrophy. The course of the disease is a slow one and the eruption lasts for months and years. Arsenic has been of a great deal of value in the treatment.

(8) Lupus Erythematosus is of two types, the discoid or localized and the disseminate or generalized. The latter is not a common form. (a) The discoid type begins as several pinhead to bean sized maculo-papules. They are pinkish to red in color and as they grow older become a deeper red, sometimes bluish red. They are covered with a dry, very adherent scale, greyish in color. They grow slowly and extend peripherally until they form definitely elevated plaques, of a bluish red color, covered with these dry, very adherent scales. There is considerable infiltration present and the patches, especially at the borders show a definite elevation. On removing the scales comedone like plugs are seen filling the gaping mouths of the sebaceous follicles. The disease attacks by preference the nose, cheeks, lobes of the ears and scalp. (b) The common form occurs as a plaque extending over the bridge of the nose onto the cheeks in the shape of a butterfly. There are few if any subjective sensations. The disease is chronic; the lesions may remain a long time without change or under treatment undergo rapid involution.

The Treatment of Cutaneous Tuberculosis

General. In Scrofuloderma in which the skin is usually secondarily involved as a result of an underlying tuberculous gland, bone or joint, the treatment is always a surgical one. In all the other forms of cutaneous tuberculosis the treatment becomes a medical problem. Both general and local therapy are indicated. The general treatment must be the same as is now in common use in treating the differnt forms of chronic tuberculosis. Fresh air, thorough ventilation of the home and sleeping quarters, bodily hygiene, sufficient rest, sunshine, much time spent in the open, and good, wholesome food are all urgently indicated.

The administration of efficient tonics is also a valuable adjuvant. They stimulate gastric secretion and aid digestion. The two best probably are the Elixir of Iron, Quinine and Strychnine in teaspoonful doses three times a day before meals and the Elixir Gentian Glycerinatum N.F. in like doses. The arsenical preparations are of decided value in the treatment of cutaneous

tuberculosis, Fowler's solution, or Liquor Arsenici Chloridi in 5 minim doses, arsenious acid in 1/20 grain doses may be given three times daily. Cacodylate of soda has been used by some physicians with success. Of the various medicinal remedies used, none has given more satisfactory results than the use of tuberculin in proper dosage. The tuberculin therapy which is fully described in the chapter on tuberculin should be followed, giving old tuberculin in minute doses at first, gradually increasing. It will not be necessary to give very large doses. The tuberculous individual's tonic dose, evidenced by a feeling of well being, when reached, may be maintained for months.

Local Treatment. The Finsen Lamp discovered by Prof. Finsen of Copenhagen, is the best means at our disposal for treating lupus. (128) The use of phototherapy in the treatment of tuberculosis of the skin marks an epoch in the treatment of this disease. The procedure is based upon the destructive effects ultra-violet light has upon the diseased cells of lupus tissue. In the use of the ultra-violet rays the lamp must be brought into close contact with the skin under pressure in order to remove from the diseased area the blood, for this interferes with the passage of the rays. The various lamps are supplied with a window of quartz which does not, as does glass, interfere with the passage of the ultra-violet rays.

The objections to this method of treatment are that in treating large areas the method is tedious, and that in treating lupus of the mucous membranes or ulcerative patches the method cannot be used because sufficient pressure cannot be exerted on these areas.

X-Rays in experienced hands have been very successful in the treatment of the disease. The Kromayer quartz lamp has also been of value. In small patches Carbon Dioxide snow can be used with benefit. Adamson (172) has recently reported very good results in the treatment of some forms of lupus with the Liquid Acid Nitrate of Mercury. The actual cautery is at times of benefit but the method of choice when it can be used is the Finsen Light.—E. A. O.

CHAPTER 33

TUBERCULOSIS OF THE EYE AND THE MUCOUS SURFACES

(A) Tuberculosis of the Eye

General Consideration. Tuberculosis of the eye is not an uncommon affection. While this organ is not particularly susceptible to the disease, any part of the eyeball or its appendages may become involved. Here, as elsewhere in the body, the disease may, especially in its incipiency, be difficult to diagnose. With the introduction of tuberculin as a diagnostic agent, inflammatory lesions of the eye of obscure origin are frequently proven to be tuberculous. (198-207)

It is a striking fact that the eye is rarely involved in a case of open tuberculosis. It is equally striking that when the tuberculous process becomes generalized, the eye, as a rule, falls prey to the disease. The eye may become the seat of a tuberculous process in an otherwise apparently healthy individual. Tuberculosis may cause inflammation of the eye by an active lesion **in situ** or by the action of the toxins of the disease liberated at remote foci. Possibly phlyctenular kerato-conjunctivitis may be an example of the latter type of disease. The microörganisms may enter the eye endogenously by way of the blood stream, or ectogenously by direct inoculation, as through an abrasion.

Predisposing Factors. The predisposing factors entering into the contraction of the disease are: (a) Age—the disease being more frequent in children; (b) Trauma—in the presence of which the tubercle bacillus finds suitable soil for its propagation; (c) Familial tuberculosis—which subjects the individual to massive doses of tubercle bacilli; (d) City-dwelling—for like reasons; (e) Tuberculosis of the other eye predisposes its fellow to a like infection.

The tunica vasculosa which consists of the choroid, the ciliary body, and the iris is the tissue most frequently invaded. The hematogenously conveyed microörganism is arrested in the delicate capillary plexuses of the chorio-capillaris layer of the

choroid, in the ciliary processes, or in the region of the major or minor circle of the iris, in which location the tuberculous nodules first appear. From these points the disease characteristically invades adjacent and foreign tissue.

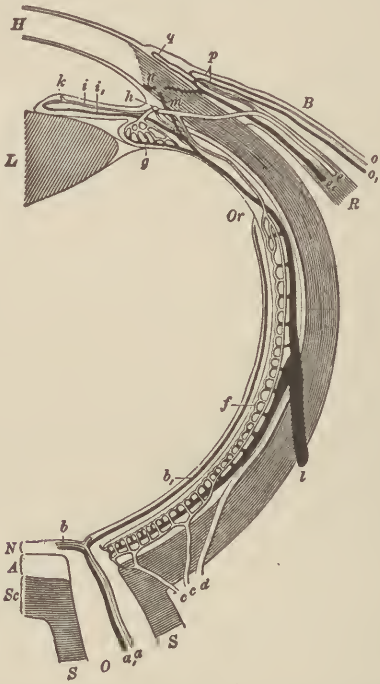

Fig. 59. Section of the Eye (Schematic) (After Leber) Showing the Usual Seat of Beginning Tuberculous Disease of the Inner Eye and in the Order of Frequency at the Points of f=Chorio-Capillaries, g=Ciliary Processes, h=Major Circle of Iris, k=Minor Circle of Iris.

Explanatory Notes

A=Chorioid, B=Conjunctiva, H=Cornea, L=Lens, N=Retina, O=Optic Nerve, OR=Ora Serata, R=Internal Rectus, S=Optic Nerve Sheath, SO=Sclera.

a=central artery, a_1=central vein, b=retinal arteries, b_1=retinal veins, cc=ciliary arteries, d=long posterior ciliary artery, d_1=anterior ciliary arteries, e_1=anterior ciliary veins, f=chorioidal capillaries, g=ciliary bodies, h=circulus arteriosus iridis major, i=arteries of the iris, i_1=veins of the iris, k=circulus arteriosus iridis minor, l=vasa vorticosa, m=veins from the ciliary muscles, n=Schlemm's canal, o and o_1=posterior conjunctival vessels, p=anterior conjunctival vessels, q=marginal loops of the cornea.

Symptoms: There are certain general clinical characteristics which usually attend the disease. They are:

1. Chronicity.
2. Relative freedom from pain.
3. The tendency to invade adjacent and foreign tissue.
4. Little response to local treatment.
5. Atypical lesions.

Since the disease may involve any part of the organ or its appendages, it may be well to briefly point out some of the more common symptoms in the several locations.

Tuberculosis of the Eyelids: Rarely seen in this country; when present, it usually is an extension of lupus of the skin. The lesions ulcerate, and the scar tissue formation following results in lid deformity. Swelling of the pre-auricular glands accompany the active lesion.

The Conjunctiva: Tuberculosis of the conjunctiva may appear as a follicular conjunctivitis, as a subepithelial nodule, or as excresences in the fornices. Either type usually ulcerates. Histologically, giant-cell systems are found. The lesions are chronic and respond but little to local treatment. The cornea may become involved and the eye-ball lost by perforating lesions.

Phlyctenular Keratitis. It has long been noted that a peculiar type of lesion of the bulbar conjunctiva occurs in children (occasionally in adults) who show evidences of tuberculous infection. The disease is called phlyctenular kerato-conjunctivitis[1] by some writers; by others, conjunctivitis eczematosa or conjunctivitis scrofulosa. These terms are suggestive of the supposed etiology. In rural districts, a history may almost invariably be obtained of an intimate association of the child with an adult suffering from pulmonary tuberculosis, whereas in cities, where such a history is usually unobtainable, the child will give a positive reaction to tuberculin. The disease very rarely occurs in nursing babies and rarely persists after puberty; adults who have not had the disease in childhood do not exhibit the affection in typical form.

These lesions are multiple, occur most frequently at the limbus of the cornea and appear as small red, superficial swellings covered by the epithelium of the conjunctiva; the vessels of the surrounding conjunctiva are engorged while the remainder of the conjunctiva may appear quite normal. The tiny tumor breaks

[1]The primary lesion, the cause of phlyctenular disease, may be a tuberculous focus somewhere in the body. This may be an enlarged gland and the advisability of its removal, if practical, should be considered or, perhaps, a roentgen ray treatment be advised. According to some authors,[89] secondary organisms are considered as playing a definite role, but how much, if any, has not been definitely established. A specific therapy has been found most suitable in connection with a general treatment as stated above. It is advisable in the beginning to give tuberculin in very small doses and in gradually increasing amounts, so as to reach a cutaneous ananaphylaxis or anergy without reaction During the treatment the skin anergy may be only transitory for with a returning of allergy or skin sensitiveness there may also be return of the phlyctenular-eczematous condition with greater virulence; hence the specific therapy demands the greatest exactitude in dealing with these lesions. The patient, during the treatment, must be under the constant care of the physician, and if any untoward symptoms appear the dosage must at once be lowered or, perhaps better, be entirely inhibited for a while.—J. R.

down at the center, producing an ulcer, which usually heals promptly. When the lesion is located on the cornea great pain, photophobia, and lacrimation accompany its cycle. The resulting desquamation may lead to a true ulcer of the cornea. These lesions are apt to appear in successive crops, continuing for months or even years. Resulting scars may disturb vision markedly, but very rarely cause blindness.

The tumors are accumulations of round cells and are in no sense true tubercles, nor have tubercle bacilli ever been demonstrated in them. It is quite probable that they are the result of protein sensitization or anaphylaxis. Possibly they may be grouped with the dermatoses known as tuberculids. The disease usually responds promptly to strict and careful regulation of the diet, general hygience, and habits of the child, cleanliness, fresh air, sunlight, and good food, with perhaps a mild specific anti-tuberculosis impression, etc., without which local treatment is but of temporary value.

The Cornea: The cornea is frequently the seat of primary tuberculosis, this from direct infection of the eye from without. Both suppurative and non-suppurative forms are seen. The suppurative forms are characterized by superficial ulcers with pannus formation. Deep infiltration or ulceration may take place leading to perforation of the cornea and loss of the eye-ball. A small percentage of the interstitial forms of keratitis may be tuberculous. The lesions are subepithelial at the beginning, frequently invading the substantia propria and leaving behind dense opacities. All atypical forms of keratitis with low grade inflammaory reaction should suggest the possibility of tuberculosis. The cornea may, of course ,become involved secondarily.

The Sclera: Tuberculous involvement of the sclera is probably always a secondary process, the uveal tract being the seat of the primary lesion. A localized, congested, tender, nodular prominence appears on the sclera which may completely disappear or which may extend and involve the cornea.

The Tunica Vasculosa: (a) **The Ciliary Body and the Iris:** Mayon, the English pathologist, removed the aqueous humor in thirty cases of irido-cyclitis and was able to isolate the tubercle bacillus in thirteen of them. The iris lesions may appear as distinct nodules, small, greyish or yellowish, most frequently situated at the root of the iris. They may be few or many. Again, there may be no visible changes in the iris, but posterior syne-

chiae form, and deposits on the posterior surface of the cornea are conspicuous. Tuberculous granuloma may form and fill the anterior chamber leading to destruction of the eye.

Tuberculous iritis may be not unlike other forms of iritis. The disease is usually characterized by little pain or inflammatory reaction and the tendency to be slowly progressive. With involvement of the ciliary body, the circumlental space may become occluded and the area bounded by the posterior surface of the iris, the lens, and the hyaloid membrane becomes filled with tuberculous tissue, while the vitreous may remain uninvaded.

(b) **Choroiditis.** The best known form of tuberculous choroiditis is the type occurring in acute miliary tuberculosis. These lesions occurring shortly before death are probably always present as a part of the disseminated process. They are grouped usually at the posterior pole of the eye, but may be peripheric. In size they may vary from one-third to disc size. In color, they are greyish or greyish-yellow, and have soft, lightly defined margins shading off into the normal retina. They are not pigmented. The retinal vessels arch but slightly over these areas. The lesions develop rapidly, new ones appearing from hour to hour; they are located in the coriocapillaris layer of the choroid and extend inwards to the lamina vitrea and outwards to the sclera. The media remain clear and the vision good when obtainable; the field is undisturbed and the retina uninvaded. Histologically, the lesions show typical giant-cell formation.

Single conglomerate tuberculous masses may develop in the choroid, pushing the retina before it with increase of tension and resulting in destruction of the eye-ball. These are late manifestations of tuberculosis and are of grave significance. Exudative choroiditis may be tuberculous and cannot be differentiated from the non-tuberculous forms.

(c) **The Retina.** Primary tubercle of the retina is rare, but tuberculosis may be the cause of vascular changes resulting in recurring hemorrhages into the retina or vitreous. The disease usually affects young adults, but may persist throughout life, giving rise to recurring extravasations of blood. The walls of the veins are usually involved, as is evidenced by a perivasculitis. The disease usually affects both eyes, and the prognosis is bad. The diagnosis is made by the use of tuberculin which must be most cautiously used lest the local reaction result in new hemorrhages.

(d) **The Optic Nerve.** In association with tuberculous meningitis and general tuberculosis, miliary lesions may effect the pial sheath, the chiasm, or optic tract. The optic nerve head may become invaded secondarily to tuberculosis of the choroid.

(e) **The Orbit.** The lower margin of the orbit is most frequently involved. A thickened area may be detected, the lids become edematous, the lesion finally shows fluctuation and breaks down. Denuded bone may be detected upon probing the wound.

Diagnosis. In the diagnosis of tuberculosis of the eye, tuberculin is of great value. The test is positive only in the event of a local reaction at the site of test, a focal reaction about the ocular lesion, and as systemic reaction. The ocular reaction manifests itself by a transient increase in the existing symptoms, i. e., increased photophobia and lacrimation, increased or new areas of infiltration of the cornea, increased ciliary injection, increased ciliary tenderness, increased cloudiness of the vitreous, increase in the size of the fundus lesions, or the appearance of new lesions. This so-called positive reaction may be followed by a negative one in which all these symptoms become ameliorated. All obscure eye lesions should suggest the possibility of tuberculosis and the case should be tuberculin tested.

Treatment. The treatment of tuberculosis of the eye presents difficulties which attend this disease in other organs. The general measures found to be useful in checking the disease elsewhere in the body should be adopted here. Locally, rest, the use of atropin when the uvea is involved and local stimulation by means of hot applications, etc., are indicated. When judiciously used, tuberculin often gives marvelous results. If the disease is detected in its incipiency, tuberculin often effects a prompt and complete cure.—G. W. R.

(B) Tuberculosis of the Mucous Surfaces. Under this caption is usually considered tuberculous disease of those mucous surfaces which can readily be inspected. This includes the tissues of the upper air passages, the ear, and the eye. The latter, however, has been already considered fully in detail; hence we shall include here only a consideration of the mucous surfaces of the mouth and nose and, perhaps, of the ear.

In tuberculous disease of such organs and tissues of the body as the intestinal canal, the lungs, the genito-urinary tract, etc., the mucous surfaces are always more or less involved in the tuberculous process; hence they are not considered here.

Tuberculosis of the mucous membranes includes tuberculous disorders found in the mouth, the nose, and occasionally in the ear. In the mouth, the disease manifests itself as affecting the lips, the cheeks, the tongue, the gums and alveolar processes, the hard and soft palate, the uvula, the faucial tonsils, the pillars, the lymphoid tissue and posterior wall of the oropharynx; in the nose, usually the septum, the turbinated bodies, and the pharyngeal tonsil of the naso-pharynx, and in the ear, the external meatus and the canal.

The mucous surfaces of all these various structures are very similar to the mucous membranes of the other parts of the body. All the surfaces offer great resistance to invading bacteria; it is only when these membranes are not intact or when the bacteria appear in too great a number, are massive, that infection can take place. Usually for infection to take place here, a slight trauma is always necessary. The secretions of the mouth and the vibrissae of the nose also offer, in a measure, protection against bacillary invasion; the mouth secretions particularly contain bactericidal substances which inhibit, destroy, or weaken bacterial implantation and growth.

Tuberculosis of the various mucous membranes may be either primary or secondary. **Primary tuberculosis** of the mouth is extremely rare; it is only through injuries to these surfaces that it is possible of conceiving a bacillary implantation. The primary form is occasionally observed, but even this is very infrequent considering the great prevalence of pulmonary disease. Primary tuberculosis of the tonsils is occasionally observed. It occurs as a result of mouth inhalation of bacilli contaminating the air currents or in the taking of food containing the bacteria; but here, also, the inhibiting action of the crypts, follicles and their secretion is distinctly noticeable.[2]

In a similar manner we can conceive of primary tuberculosis of the pharyngeal tonsil by the inhalation of air through the nasal passages. Microscopical examinations of tonsils after tonsillectomy has often revealed the presence of the tubercle bacillus as a latent process, but tuberculous disease of the tonsils, is not frequently found. It seems that the bacilli pass through the tonils as through a sieve, and we find them in the regional, the cervical glands, which then frequently become tuberculously dis-

[2]Bacilli do not readily enter the faucial tonsil; the epithelial covering (even within the crypts) seems to form a safe barrier. It is only through a massive infection that this protective mechanism becomes weakened and its inhibitive functions lost.

eased and undergo degenerative changes. The portals, (the tonsils) may remain enlarged, but as a rule no other noticeable changes are observed. We can also readily conceive primary or direct infection in the non-tuberculous by the use of an unsterilized tooth forceps immediately after the removal of a tooth from an actively tuberculous individual, and that can also apply to nasal or aural operations through unclean instruments.

The Secondary Form of Tuberculosis of the mucous membranes is occasionally observed in the actively tuberculous when the sputum is bacilli laden, where, for instance, the removal of an offending tooth may be followed by a tuberculous ulceration about the gums and the deeper structures in and about the alveolar process. Tuberculous ulcers about the tongue, cheeks, mouth, nose, or ear in tuberculous subjects are occasionally observed; these usually are secondary to slight trauma or abrasions of the mucosa. Such ulcers are usually superficial, flat, pale, of irregular outline, covered with dirty, grayish-white secretions, generally situated upon a nodular base without inflammatory border, and covered with red granulations interspersed throughout by grayish-yellow spots. The whole process is a slow one, advancing slowly and healing slowly. That these ulcerative surfaces are of a tuberculous nature is evidenced from microscopic sections in which we find collections of round cells interspersed with giant cells and occasionally tubercle bacilli. These ulcerative processes are extremely chronic, accompanied by little or no pain, and may exist without subjective symptoms for some time before the patient becomes aware of their presence. This is particularly observed if the lesions are situated about the lips, gums, or tongue; not so, however, if on the pharynx where during the act of swallowing they become extremely painful. Tuberculous ulcers following the removal of a tooth[3] in an advanced case of pulmonary tuberculosis is always accompanied by most excruciating pain, and here the ulcerative process usually invades the whole alveolar process. Tuberculosis of the mucous surfaces is more frequent in men than in women in proportion of two to one; this is because men are

[3]A most distressing case of tuberculosis of the mucous membrane of the mouth, which typically reflects what has been stated above, came under my observation recently. A tuberculous individual, third stage, far advanced, but still ambulatory, his sputum laden with tubercle bacilli, had five teeth removed, three from the upper jaw and two from the lower. Almost immediately, this was followed by tuberculous ulcers which invaded the entire depth of the alveolar processes. Owing to his poor physical condition, these ulcers spread with great rapidity over much of the surface of the jaws and gums. The time from the removal of the teeth until exitus was only two months, but during all that time he suffered most excruciating pain and distress unless relieved by anodynes.

more careless about the hygiene of their mouths, picking their noses, putting the fingers in the ears, etc.

Many of these ulcerative tuberculous processes are lupoid growths, if not typical lupus, and most are akin to the tuberculids found in cutaneous lesions. Owing to their very slow growth they may resemble at first only small nodules or tumors which gradually undergo degenerative changes with tissue destruction, when their true nature becomes revealed.

The tuberculous processes about the nose are usually observed in two forms; one an infiltrative, ulcerative form and one of tumor formation. The septum here is frequently primarily infected, but may be secondarily from the bacilli laden sputum of the tuberculous individual.

The external ear and the auditory canal may also be the seat of tuberculous disease, this is usually a secondary form, a finger infection.

Treatment. Contemplated operations about the mouth, nose, eyes, or ears of an actively tuberculous individual should receive the closest consideration and if about the nose or mouth should be undertaken only when we are assured that the sputum is entirely free from the Koch's bacillus. Even under the most painstaking cleanliness and a strictly aseptic technic it is almost impossible to avoid wound infection and owing to the extremely great tendency of the ulcerative process to spread following operative interference, cauterization, or curettage, it is advisable to be most cautious in the use of these measures. A general treatment, if not already instituted, must be secured at once. This is just as important here as it is in treating tuberculosis in other parts of the body, and should be done in connection with topical applications of appropriate remedies which tend to relieve pain and suffering. The use of a 2 to 5% cocaine solution applied directly to the ulcer after it has been freed from secretions often gives prompt relief. Morphine sulphate may be used similarly, but is better given by mouth. A saturated solution of pyoktanin blue applied directly to the ulcer, the use of iodine in the form of the tincture, or anesthesin or orthoform in powder form applied directly is often followed by most gratifying results. Lactic acid, applied and repeated in about three hours, if carefully used, is often very serviceable.—J. R.

CHAPTER 34

TUBERCULOSIS AND THE CARDIO-VASCULAR SYSTEM.

1. **Tuberculosis and the Heart.** It goes without saying that a cardiac lesion may influence mechanically a tendency to disorder in the pulmonary circuit, or, if the lungs are already diseased, to favor still more the disturbance. In practice, we usually observe two conditions, namely, either a stenosis of the pulmonary artery or a valvular lesion of the left heart. Pulmonary stenosis as a factor in tuberculous disease is now generally recognized, and individuals with a stenosis of the pulmonary artery, which is usually congenital, generally perish when they reach the age of puberty or about that time. In valvular lesions of the left heart, however, accompanying a tuberculous process, the disturbance is generally of mechanical origin, a faulty lymph and blood circulation. In some individuals, there is always a strong tendency to a mechanical disposition to blood and lymph stasis which favors a lessened excursion and aeration of the apices of the lungs, a disposition to tuberculosis. (135)

Rokitansky, in 1846, promulgated an antagonism between pulmonary tuberculosis and disease of the left heart, namely, that within certain limits, a primary cardiac disease would inhibit the co-existence of a secondary pulmonary tuberculous disorder. Since Rokitansky's time, many observations have been made by competent observers, and they have shown that both aortic and mitral disease may accompany pulmonary tuberculosis.

Fremmolt, in 1874, reported on 8,000 autopsies, and showed that not infrequently there was a simultaneous appearance of cardiac disease and pulmonary tuberculosis. He arrived at the following conclusions: (1) The simultaneous appearance of chronic, cardiac disease and pulmonary tuberculosis is not as infrequent as is usually assumed. (2) Disease of the aortic valve (the left ostium arteriosum) is found to complicate pulmonary tuberculosis somewhat less frequently than affections of the mitral valve, (the left ostium venosum). (3) The simultaneous disorder of more than one valve is extremely seldom.

Kryger, in 1,100 autopsies, frequently found healed pulmonary lesions in cardiac disease, and he concluded that a tuberculous process is seldom an accompaniment of cardiac disorder. Traube maintained that tuberculosis may exist with aortic lesions, but not with mitral stenosis. Lyden joined Traube in the belief that mitral stenosis and pulmonary tuberculosis do not occur simultaneously, but he also emphasized the infrequency of both mitral and aortic insufficiency with tuberculosis. Squire and Frankle, on the other hand, observed that pulmonary tuberculosis and cardiac disease are frequent, both with mitral stenosis and other cardiac lesions. Fossier states that both disorders in the same individual are not infrequent, particularly with mitral disease, and that tuberculosis may develop at any stage of the cardiac difficulty; further, that in ordinary pulmonary tuberculosis, the heart may be smaller, but when complicated with cardiac lesions there is no atrophy; if tuberculosis is present it may progress, independently of the cardiac disorder, sometimes slowly, sometimes more rapid, when the prognosis becomes more grave, the dyspnea greater and although the tuberculous process is not influenced by the cardiac disorder still it exerts a most unfavorable influence on the heart.

Meisenberg in Curschman's clinic found, in 4649 tuberculous individuals, 53 cases of co-existing heart lesion (1.14%), and mitral insufficiency was the most frequent single lesion. He further observed that the pulmonary tuberculous process influenced the cardiac disorder unfavorably, but was in turn little, if at all, influenced by the heart lesion. In 33% of his cases, the cardiac lesion was the older disorder; in 4 cases, the tuberculous process was undoubtedly the older, and in the remaining cases it was most difficult to determine which was the primary disease. In none of his cases, however, was mitral stenosis the older lesion. Sauer, Berlin Pathological Institute, in 304 carefully selected mitral disorders, found 85% entirely free from tuberculous disease, in 12% a healed lesion, and only in 3% an accompanying active pulmonary affection, and all of his cases showed a clinical induration, which confirmed the belief that indurated tissue does not offer a very favorable nidus for either the deposition or the growth of the tubercle bacillus.

It is evident that it becomes necessary to know which was the primary difficulty, the cardiac or the pulmonary, and it also becomes evident that to a primary, perhaps already somewhat ad-

vanced, pulmonary tuberculous process, a cardiac lesion may be added secondarily, or the conditions may be most favorable, (in mixed infection and in ulcerative processes in the lungs), to bring about a cardiac disturbance. Hence from all that has been stated we may conclude that (1) Rokitansky's theory of an antagonism existing between tuberculosis and cardiac disease is not tenable; (2) mitral stenosis is less frequently observed in combination with pulmonary tuberculosis than are the other valve lesions; (3) mitral insufficiency may bring about a certain degree of immunity in tuberculosis when it favors the development of the brown induration in consequence of pulmonary stasis; (4) the progress of an existing tuberculous process will be neither modified nor inhibited by a co-existing cardiac disease, but (5) the tuberculous process reacts very unfavorably on the cardiac disorder the more rapidly the tuberculous process advances.

The fact remains that there exists a lessened disposition to the deposition and growth of the tubercle bacilli in mitral stenosis. This is supposed to be the result of blood stasis in the lesser circulation, but there exists no stasis in well compensated mitral stenosis. Only with the appearance of decompensation does slowing of the blood current and stasis supervene; hence, the lessened disposition to the deposit of the tubercle bacilli can, according to v. Romberg only be explained on a mechanical basis, namely, that in mitral stenosis the smaller blood vessels in the pulmonary circuit are much distorted this lessens the mechanical opportunity of the tubercle bacilli, which are usually in clumps, forming small emboli ready to be deposited. In mitral stenosis, stasis is greater than in insufficiency. It may be noted here that hypertrophy of the left ventricle and pulmonary tuberculosis are also antagonistic, that in aortic valve lesion and in arterio-sclerosis there is usually pronounced hypertrophy of the left ventricle. These conditions are generally found in advanced age when clinical tuberculosis becomes more infrequent.

2. **Tuberculosis and the Pulmonary Circulation.** The anatomical condition of the heart of the tuberculous individual has early been recognized. It has generally been described as small[1], both

[1]The small heart in the tuberculous. Heart proportional to body weight. Animals like cattle, antelope, etc., in which the heart is in proportion to the body weight as 1 is to 250 are very prone to tuberculous disease, whereas the sheep, whose proportion is as 1 to 100, the deer, 1 to 90, are comparatively immune. In the human, an enlarged heart is extremely seldom found in the tuberculous subject, and in the gouty or podagric patient, who usually has an enlarged heart, tuberculosis is unknown as a concomitant disorder.

in size and in the thickness of its walls as well as the capacity of its ventricles. Peacock found the heart in the tuberculous smaller than that found in other chronic diseases, and Louis, Lännec, Stokes, and Rokitansky all speak of the small heart in the tuberculous which they attribute mainly to nutritional disturbances, to a lowering of the general muscle tone, the cachexia and the lessening of the quantity and quality of the blood. Engel demonstrated that the left heart becomes proportionately less in weight as compared with the right. Normally the right heart is to left as 1:2.82; in the phthisical, however, the right is to the left as 1:2.14.

Of much importance to the theory of the small heart of the consumptive is the teaching of Benecke, namely, that with the small heart, the lumen of the arteries becomes smaller, causing a lessening of the blood supply through the pulmonary circuit and a weakening of the lung structure, either an inherited or acquired disposition. A primary constitutional anomaly favoring the tuberculous disease must be presupposed, the analogue of which is found in Virchow's teaching of the inherited hypoplasia in the arteries of the body as found in the thinness of the walls and in the lessening of their lumen as the pathogenesis in many cases of severe chlorosis. Brehmer pointed out that with a small heart, if the lung is too large, a relative anemia of the latter results, and, with a lessening of the propelling force, a mechanical predisposition to tuberculous disease is established.

Decroix and Potain consider the small heart in the tuberculous a secondary process due to the cachexia induced by the malady. According to Müller, the small heart in the phthisical is not a congenital hypoplasia, but a progressive decrease in weight which runs parallel with the general emaciation of the whole body, particularly that of the muscular structure. The roentgen ray was called into use by Moritz and by exact orthodiagraphic methods, he found, in a most remarkable degree, that the size of the heart is a very changeable factor, dependent upon the size of the body of the individual, on the sex, the age, the conformation of the chest, and, above all, upon the muscular development and the weight of the body. Sciallero, from an analysis of thousands of radiological plates, found the heart in the young suffering from malignant disease always small; in people somewhat advanced in years, however, or in those suffer-

ing from inactive or arrested tuberculous disease, it was almost always normal and small only in few exceptions.

Beck studied the relation of the body weight to tuberculosis and concluded that the volume of the heart as compared to the body weight becomes lessened. Kraus pointed out that the small heart, situated centrally, with large vessels is the typical heart of the contracted chest or the phthisical habitus. This all goes to prove that the heart of the tuberculous shows at autopsy a lessening in cardiac volume due to a cachexia. Landouzy describes a "paratuberculous" condition, said to be noticeable in the heart and blood vessels of the descendants of the tuberculous, which may be due to a congenital smallness of the thoracic cavity. It has not, however, been definitely proven that a small heart is really a predisposing factor to tuberculous disease. If we assume a relative pulmonary anemia necessary to a tuberculous disorder, we must first prove that a small heart is really incapable of supplying the needed blood to the lungs.

Much interest has always been exhibited in the relationship of tuberculosis to the right heart, and Buhl definitely proved the appearance of an eccentric hypertrophy with dilatation of the heart in tuberculosis. In rapidly progressive cases, frequently, dilatation of the right heart is observed, particularly in chronic phthisis and in the fibroid variety. It has long been known that certain complications lead to enlargement of the right heart, and pleural adhesions, bronchial catarrh, and emphysema may be mentioned; complications in pulmonary tuberculosis, however, like chronic bronchitis, pleural adhesions, and fibrous induration of the pulmonary tissue, and, especially, emphysema all play a characteristic role in the dilatation of the right heart. Hirsch, following the views upheld by Müller, demonstrated that hypertrophy of the right heart in tuberculosis is based mainly on mechanical changes in the pulmonary circuit, that is, from pleural adhesions, from emphysema, and, in a number of cases, from the contractions of the arterial lumen due to indurative or ulcerative, tuberculous processes.

Dilatation of the right ventricle and accompanying enlargement of the liver have only recently been advocated as early symptoms of pulmonary tuberculosis. It should here be mentioned, as was first related by Turban, that an absolute cardiac dulness toward the right of the sternum is a very common and an early symptom of a right-sided apex tuberculosis with con-

traction, and this retraction of the lung border towards the right may leave the right heart exposed and thus may lead to error in the diagnosis of a cardiac hypertrophy or an enlargement towards the right. That we may assume a frequent hypertrophy of the right ventricle in tuberculosis is evidenced by accentuation of the second pulmonic tone.

The heart may further be influenced by the tuberculous processes in various ways, namely, by dislocation and distortion, an interference with the functions of the heart, causing both subjective and objective symptoms to be present. Cardiac palpitation, abnormally loud heart sounds, pain, pulse rapidity, etc., are frequently observed as a sequence of cardiac dislocation, resulting from a tuberculous process as in apical fibrosis, an exudative pleurisy or a pneumothorax, and these symptoms are clearly the result of mechanical and not of toxic disturbances.

Chronic changes in the heart structure, the endocardium, the myocardium, and the pericardium as well, have frequently been observed as complications in tuberculosis. Neuman, in 1,489 autopsies on phthisical subjects, found five cases of chronic, interstitial, fibroid myocarditis, and Norris, in 41 carefully selected cases of phthisis, found in 14 the heart muscle histologically normal, in 10 chronic, interstitial myocarditis, in 5 fatty degenerations, and in 3 brown induration and atrophy. Fatty or muscle degeneration have very frequently been described by pathologists as an accompanying disorder in chronic phthisis. Teissier emphasizes the frequency of the tuberculous etiology in many of the disorders of the valves of the heart. This is analogous to the teaching of Poncet that a tuberculous joint rheumatism, an arthritis, or an arthralgia is the result of the toxin action and not of bacillary origin. It is the bacillary toxins (the tuberculin) circulating in the blood of the tuberculous which cause the change in the heart structure, both endocardial and myocardial.

The heart alone is not influenced by the tuberculous process, the blood vessels also show the effect of the disturbance. We frequently observe in the tuberculous, thrombosis of veins, the direct effect of a bacillemia, the arteries are also frequently affected by the tuberculous process, and even arterio-sclerosis is very often found as a concomitant symptom in young phthisical subjects.

From clinical observation and study of pulmonary tuberculosis in its relation to the heart since Rokitansky's time, the following deductions may be made. The heart in the phthisical may be normal and be displaced, more often to the right, either from pleural adhesions or exudates, or from chronic, fibroid, tuberculous changes in the lungs. The displacement may be either sudden, as in natural pneumothorax or serous pleurisy, or it may be pushed to one side by a distended lung and drawn toward the contracted lung where often it may simulate a dextrocardia. In the beginning of the tuberculous process, the heart is usually normal in size, but with the progress of the disorder some changes are more or less noticeable, and the heart becomes atrophied or, occasionally, hypertrophied. This hypertrophy is especially observed in cases of old and chronic, fibroid phthisis in which a cardiac insufficiency accompanied by alarming symptoms is noticeable.

In about 60% of the cases of pulmonary tuberculosis, auscultatory heart changes are demonstrable. In advanced pulmonary tuberculosis, accentuation of the pulmonic second sound has been long observed and functional murmurs are heard in about 6% of all cases. The pulse is usually of low tension and rapid. Of all organic heart lesions, a pericarditis is the most frequent complication. This, however, may be tuberculous or non-tuberculous, more often of the dry than of the exudative form. Myocarditis is infrequent and usually non-tuberculous. A tuberculous endocarditis is very infrequent; in fact, endocardial disease in the course of tuberculosis is very seldom, although an individual suffering from endocarditis may be stricken with pulmonary tuberculosis. Pulmonary stenosis is always primary, mitral and aortic insufficiency generally primary, but mitral stenosis may be secondary. Mitral insufficiency is the most frequent cardiac disturbance encountered as a complication in pulmonary tuberculosis, and mitral stenosis is the least. Aortic insufficiency is also frequent, whilst aortic stenosis is very seldom. With the exception of pulmonary stenosis, a cardiac lesion appears to inhibit or lessen the tendency to tuberculous disease.

3. Tuberculosis and Tachycardia. An organ in which the anatomical structure is subject to many changes must suffer in its proper functioning, and one of the first and most important observations made is that of the disturbance of the heart in tuberculosis. The rapid action of the heart, the accompanying

tachycardia, is a phenomenon resulting from the mechanical changes in the lesser circulation, and the more pulmonary tissue that is involved, the more rapid will be the heart's action. Often in chronic cases where the process is arrested, even after many years, or in cases of extensive fibrosis or pleural thickening, slight exertion will bring about a rapid heart action. It is very evident that even in recent cases of pulmonary tuberculosis with extensive involvement a frequency of the pulse can be explained on mechanical grounds. A tachycardia which may appear independent of fever usually runs parallel with the severity of the disorder. Sterling mentions that a rapid pulse in the first stage of the pulmonary disorder proves the absence of a connective tissue wall about the tuberculous process.

The clinical symptom of tachycardia in tuberculosis is the extremely abnormal lability of the pulse, and this lability remains for many years after all the symptoms of the pulmonary disorder have disappeared. This rapid heart action, even after complete inactivity, points to a remaining slight injury to the heart muscle or to the nervous apparatus of the heart. A constant, rapid pulse is one of the earliest symptoms of the tuberculous disorder, and its constancy is of the greatest prognostic importance.

Tachycardia in the tuberculous may be due to pressure upon the nerves of the heart such as accompanies enlargement of the tracheo-bronchial glands or to mediastinitis, pleuritis, pericarditis, etc., in all of which the heart's rapidity is more or less due to mechanical causes. It is possible, although improbable, that, in the tuberculous, compression of the vagi is followed by tachycardiac; theoretically, compression is usually followed by lessening of the pulse rate. Animal experimentation has shown the effect of severing the vagi, namely, that the severing of one has no effect on the heart's action, but if both are severed the frequency of the pulse rate increases. It may be assumed that in tachycardia the fibres of the sympatheticus are compressed with the fibres of the vagi. In most instances the pulse remains always fast.

Lawrason Brown called attention to the frequency of an auscultatory phenomenon observed in the heart of the tuberculous, notably, an impurity of the first mitral tone and a systolic murmur at the apex. The possibility of a slight dilatation of the heart as the cause of this systolic murmur has first been men-

tioned by Fürbringer. The accentuation of the second pulmonic in tuberculosis as a sign of pressure in the lesser circulation and of hypertrophy of the right heart has already been mentioned.

4. **Tuberculosis and Hypotension.** A low tensioned pulse in persons of middle life is not a constant phenomenon in the tuberculous individual, although hypotension is the general rule, a normal or high pressure is a favorable sign, as is a rise in pressure during the course of the disease. In rapidly progressive cases, the blood pressure is low and remains low. Blood pressure observation is one of the surest and safest signs to guide us and to point out the cases which can be healed or be arrested. These observations apply not only to pulmonary tuberculosis but to tuberculous pleuritis and peritonitis as well.

In beginning tuberculosis, the blood pressure may be within normal limits, but in the third stage cases it is usually low; this applies most particularly to those rapidly progressing cases with cavity formation. Observation has proven that in progressive tuberculosis there is a proportional lowering of the pressure—this lowering of the pressure depends upon the activity or non-activity of the tuberculous process at the time. The explanation of this lowering of the blood pressure in tuberculosis is chiefly the result of changes in the muscular structure of the heart brought about by the elaborated toxins of bacillary origin and vaso-dilator action by the same bacillary products.

With the lowering of the pressure, the rise in the frequency of the heart action goes hand in hand. This is due to a paralysis of the vaso-constrictors in consequence of which the frequency of the heart's action is quickened in order to equalize the paretic condition in the circulation. In some supposedly normal subjects, the use of tuberculin has been followed by a lowering of the blood pressure, which generally reaches its minimum point in about eight hours, and returns to normal again in about three days. A logical deduction of the low tension in the phthisical is supposed to be based upon the hypofunctioning of the adrenals. Observations along this line have been made by Bignold, Lucien, Bernard, Löschke, and others, all of whom have observed an inhibition of the proper functioning of these glands in tuberculous subjects. Tuberculous individuals with a favorable[2] prognosis

[2]It is a more favorable prognosis when the arterial tension in the tuberculous individual is found normal or above. A sudden rise in the pressure may be a forerunner of an impending hemorrhage and a sudden drop a sign of an acute or recurring exacerbation or perhaps the beginning of a generalized disease. The low blood pressure in the tuberculous may be explained by the presence of tuberculo-toxins in the circulation.

generally show a normal or high blood pressure, and if, in patients suffering from arterio-sclerosis or nephritis, tuberculosis becomes a concomitant disorder the blood pressure will always remain high. It is a notable fact that in the tuberculous threatened with hemorrhage, or perhaps some days or weeks before the onset of the hemorrhage, the blood pressure usually becomes high. Neuman observed this in more than 86% of his cases.

5. Blood Pressure Observations.(74) It is now generally conceded that pulmonary tuberculosis as clinically observed is usually accompanied by a marked diminution in the blood pressure, and that this lowering of the tension is ascribed to a toxemia, to the circulating in the blood-stream of various toxins of bacillary origin, and that the development and elaboration of the toxins during the progress of the disease and subsequent entrance into the circulation in progressively increasing amounts will induce irritation and dilatation of the arteries, a vasomotor irritability, and subsequent paresis, and attendant diminution in the blood pressure.

That tuberculin, the toxin elaborated during bacillary growth. has a definite influence on the circulation and on the arterial tension and blood pressure has been amply demonstrated by F. Bauer, of Vienna, who, after repeated injections of tuberculin, and which reacted negatively, observed no variation in the blood pressure; if, however, the injection was followed by a positive reaction, at least a slight, sensible lowering of the pressure was always observable. The direct effect of the toxemia is to cause a rapidity of the heart's action, accompanied by a weak pulse, a want of arterial tone, resulting in diminution of the internal pressure; hence, there is a corresponding lowering of the blood-pressure with the progress of the pulmonary lesion and attendant toxin absorption, and, inversely, with a lessening of the active tuberculous process, lessening of toxin elaboration and absorption, a corresponding rise in the blood-pressure may be demonstrable.

In most tuberculous patients, a distinct variation in pulse and tension is always clearly observable, whereas in a healthy individual a very slight variation in pulse-rate and blood-pressure may be noticeable. The pulse-rate is, in health, somewhat lower while in the recumbent position, higher in the upright, and at a point midway between the two in the sitting, with a corresponding rise of the blood-pressure in the horizontal, a lowering of

pressure in the vertical position registering a pressure between the two, in the sitting posture. In the tuberculous, there is always noticeable the great extremes both in the pulse-rate and the blood-pressure observations. In taking the blood-pressure and pulse-rate of tuberculous patients in the three stages of the disease, nothing more striking can be demonstrated than that usually the arterial tension is lower and the pulse-rate higher in the actively progressive cases than in the chronic or arrested.

From blood pressure observations Pottenger has formulated the following:

1. In advanced pulmonary tuberculosis, the blood-pressure is low.

2. Causes which favor this low blood-pressure are: (a) Toxemia—the action of the toxins on the vasomotor dilators; (b) weakness of heart muscle, and (c) general emaciation.

3. Causes which maintain a proper amount of tension are cardiac hypertrophy and thickening of the arterial walls.

Nardi, of Padua, who thoroughly studied but a limited number of cases, 30 in all, arrived at like results. Of the 30 cases, 9 showed a lowering of the tension; 12 were within normal limits, and 9 showed a hypertension. He divides all tuberculous cases into two groups: The first group, the *energetic tuberculous* with normal or overnormal pressure, a relatively large heart, a better development of the left ventricle, a good body development, a good resisting power, and a better prognosis. The second group is designated as the *torpid tuberculous,* with a pressure below normal, a relatively small heart, positive clinical picture, poor resisting power, and an unfavorable prognosis. He maintains that the prognostically unfavorable blood-pressure lowering in a certain class of the tuberculous is the direct result of the faulty development of the heart, reviving Rokitansky's theory of the small heart in the phthisical.

In many of the suspected tuberculous in which the clinical picture points strongly to a pulmonary tuberculous involvement, even before a physical examination is made, a fair diagnosis can often be made by blood-pressure and pulse-rate alone. With a fairly rapid but weak pulse in the lying, faster in the sitting, and still faster in the standing posture, a pronounced tachycardia, and an arterial tension which is best with the patient in the recumbent, not so good in the sitting, and much poorer in the upright position, a fairly positive diagnosis of pulmonary tuberculosis

may be supposed. If to this be added a rise in temperature, loss of weight, of appetite, cough, and like symptoms, a positive diagnosis from such blood-pressure findings and pulse-rate records can often be made tentatively without physical examination of the chest.

In taking the pulse-rate and blood-pressure of 74 ambulatory cases at the tuberculosis clinic at Rush Medical College, representing the incipient tuberculous, the moderately advanced, and far advanced, as well as tentative and non-tuberculous, quite a difference in results was tabulated. In the records of the 32 first-stage cases, representing the ages from sixteen in the youngest to thirty-six in the oldest, all taken in the three varying positions, one is impressed by the frequency with which the pulse-rate stands in inverse proportion to the blood-pressure. This variation cannot be referred to as a mere coincidence. With a gradual rise of the pulse first from the lying to the sitting, then to the standing position, there is a proportional fall in the blood-pressure which is not in correlation with healthy records. If there be a rise of 10 beats from the lying to the sitting, and probably 10 more from the sitting to the standing position, there will be in many cases a corresponding drop of about 6 points in the blood-pressure from the lying to the sitting, and 6 more points from the sitting to the standing position. In the 14 moderately advanced cases the inverse difference between the pulse-rate and the radial tension was still more noticeable in many of the cases. In the advanced, old, chronic cases with much fibroid tissue changes and good resisting power, the variation in the pulse-rate in the three positions—lying, sitting, and standing—may be as great as in the early infected, active cases, but a decided difference in the blood-pressure was observable. Here a variation in pulse-rate in lying, sitting, and standing of 90, 102, and 110, respectively gave a uniform blood-pressure of 120 in assuming either lying, sitting, or standing position.

Summarizing:

1. Blood-pressure and pulse-rate in the tuberculous is only a symptom, and not a part, of the disease.

2. Lowering of the blood-pressure may be interpreted as an early symptom of the tuberculous infection, even before the physical signs or even before elevation of temperature may be definitely demonstrable.

3. The toxemia in the tuberculous, with its accompanying cardiac atrophy and degeneration, seems to favor hypotension and tachycardia, concomitant vasomotor dilatation, and a low blood-pressure, whereas even with a moderate toxemia in the tuberculous, with a relatively large heart, a better development of the left ventricle will favor normotension or hypertension, and consequently a high blood-pressure.

4. Tuberculous patients with high blood-pressure, even though the cause of the increased pressure is an arteriosclerosis, primary or secondary to a chronic nephritis, do better than patients with subnormal or hypotension (Haven Emerson).

5. In the tuberculous subject suffering from vasomotor dilatation induced by the circulating toxins in the blood, anything which will cause a contraction of the arterioles will increase vascular tension; hence, vasomotor pressor remedies may be given, the first and foremost of which is rest.

6. Lowering of the blood-pressure in the active tuberculous may be a conservative act of nature—may be compensatory. Is it advisable to raise the pressure by the administering of blood-pressure-raising drugs? Would it be beneficial or harmful?

7. A favorable prognosis may be expected if, during the treatment of the tuberculous, the blood-pressure obtains a steady and uniform rise, approaching nearer the normal as the disease is being arrested.

8. The taking of the blood-pressure on the tuberculous patient in but one position is faulty, and will not lead to proper deductions, consequently a change of position from the lying to the sitting and to the standing should be made while taking the pulse-rate and making proper blood-pressure observations.

Early recognition of the flaring up of a latent or a quiescent focus is diagnosing early or beginning tuberculosis. In beginning tuberculosis the blood-pressure is lowered, the pulse-rate increased; in the second stage the pressure is lowered more and the pulse frequently increased. In the young and in the female the blood-pressure is higher proportionately than in men and in the aged. The pressure is increased by excitation and by cough and is lowered by exhausting labor or after a full meal. A rise in blood-pressure is prognostically a favorable sign in one in whom the pressure was previously low, and remaining low indicates a progression of the disorder. Low pressure is charcateristic of and constant in the active tuberculous, hypotonia is the rule. By means of blood-pressure notation the curable can be separated from the non-curable. Usually in cases with a tendency to cavity formation or the breaking down of tissue the pressure is low, is labile, not stabile. More than one reading is necessary.

CHAPTER 35

TUBERCULOSIS FOLLOWING TRAUMA AND SHOCK

The Relation of Trauma and Shock to Pulmonary Tuberculosis.

The Municipal Tuberculosis Sanitarium (Sanatorium) of the City of Chicago was completed and opened for the reception of patients in April, 1915. From the date of opening of the Sanatorium until the end of the year, that is, until December 31, 1915, covering a period of about nine months, the Central Free Dispensary of Rush Medical College admitted to the Sanatorium through its channels 257 patients. In looking over the records and histories of these 257 tuberculous individuals it was found that about 77 (30%) gave a history of having had an operation performed within a year or so previous to the onset of active tuberculous disease. The question now arose: Was the frequency of active pulmonary tuberculous disease in these individuals who had submitted to operation a mere coincidence or was the giving of the anesthetic, the shock incident to the operation or the operation itself a contributing factor in the etiology of tuberculosis? It is not difficult to conceive that an operation attended by loss of much blood, owing to lowering of vitality may activate a previously latent tuberculous process, or that the administering of an anesthetic is too concentrated a form, say, ether in more than 8%, tuberculosis may be brought about. However, considering the question of shock as a cause of tuberculous disease the problem becomes much more complicated. Regarding the question of shock as a factor, we must consider: (1) the definition and what are the causes of shock; (2) what are its signs and symptoms and (3) what influence has shock, and trauma as well, upon the quiescent tuberculous process so as to reactivate it?

(A) What is the definition of shock and what are its causes? Shock is prostration, is collapse, is a profound stupor and is described as a sudden physical or mental disturbance, a state of profound mental or physical depression consequent upon severe physical injury or emotional disturbance. It may be brought

about by any injury to either body[1] or mind, such as fear, grief. anxiety, worry, homesickness, even great joy, toxic drugs, like mercury, arsenic and tobacco, extremes of heat and cold, despondency, mechanical injuries to vital organs, the brain, stomach, viscera, testicles, ovaries, skin, joints, extensive burns, etc., and all severe pain capable of exhausting the vital powers. Some individuals predispose to shock, the sanguine temperament more frequent than the lymphatic, the overworked, the underfed more than the idle, the anemic and cachetic more than others. There may be various degrees of shock and the injury to one organ followed by greater shock than to another. In intestinal trauma the nearer to the pylorus the injury the greater the effect of nervous shock. Iliac obstruction in the dog may be followed by death after weeks but if duodenal, death may follow in a few hours. Shock is frequently induced by surgical operations, in all of which we recognize a traumatism to either body or mind. Trauma and shock are not synonymous; they are interrelated. interdependent, no trauma, no shock; shock follows trauma, hence tuberculosis following shock presupposes a previous mental or physical injury.

(B) What are the signs and symptoms of shock? Trauma is always presupposed to shock. the individual suffering from shock presenting a state of extreme torpidity. Apathetic, face and skin pale, lips and nails cyanotic, clammy perspiration, pupils dilated. eyes staring, with a fixed look into the distance, nausea, vomiting, hiccough, respiration accelerated, irregular, respiratory wave shortened, both inspiration and expiration are quickened and the pause lengthened. Blood pressure and pulse are most interesting and pathognomonic. In surgical shock, the abnormally low blood pressure is the most essential, single phenomenon, the arterial pressure may be reduced to or even below 50 mm of mercury. A noted lowering of the blood pressure is often observed immediately on incising the skin of the abdomen. There is usually a pronounced reduction in body temperature, may drop from 1 to 3 degrees and lowering of the temperature below 96.8 is a very grave indication; pulse weak, and not palpable. The fall in temperature in shock is the result and not the cause of the low blood pressure. In profound shock the bulk of

[1]It has repeatedly been proven that many persons harbor within their bodies virulent tubercle bacilli but are not made sick by their presence in the body. In such individuals it is only necessary that disturbances or lowering of vitality, much physical and mental efforts, trauma and shock, weakening of the body by intercurrent disease, etc., to lead from a tuberculosis infection to a tuberculous disease.

blood does not circulate freely in the arterial system but accumulates in the venous trunks resulting in intravenous hemorrhages or in symptoms which simulate hemorrhage. Shock causes a vasomotor dilatation, a lessening of vasomotor tone a result of reflex paralysis or exhaustion of the vasomotor centers, an inhibitive action of the sympathetic fibres of the vasomotor nerves, causing lowering of the blood pressure, the blood escaping into the veins and capillaries. The more highly organized the more prone to shock and a high strung, nervous temperament constitutes a most important predisposing cause. Shock is light in children, pronounced in the adult and grave in the aged. In shock all depends upon the circulatory apparatus and not the age of the patient. In the aged the arteries are hard and fibrous and the blood pressure usually high; in the young the arteries are soft and pliable and the blood pressure low. The new born is free from shock that is before the physiologic connection between the central nervous system becomes established. In the new born there is a short period of immunity which is followed by the greatest period of susceptibility which gradually lessens until in adult life the resistance is greatest and again diminishing in old age. There is little difference in the sexes before puberty; after puberty, the female becomes less stable, especially at the menstrual period, the male growing more hardy. From puberty to the child bearing period the female is slightly more susceptible to shock, the female is more hopeful, more tractable as a patient.

(C) What influence has trauma and shock upon the tuberculously infected individual? It is now generally admitted that there exists a close relationship of tuberculosis to accidents;[1] to trauma, to shock, but to bring about tuberculous disease in shock both trauma and bacilli are necessary. The view is held by many that an organ or tissue in order to become tuberculous, it must contain tubercle bacilli previous to the injury or be already tuberculously diseased. It has been repeatedly proven that tuberculosis may develop in an organ entirely free from bacilli previous to injury. Virchow and Rokitansky assumed that muscle fibre possessed a specific immunity against tuberculosis; however, animal experimentation has demonstrated that if in an animal the muscles of the extremities be bruised, this then followed by an intravenous injection of virulent bacilli that in addition to the pulmonary tuberculosis a notable tuberculosis of the irritated muscle fibre is present.

With an injury tubercle bacilli may enter the body or wounds become directly infected, and direct infection from without, exogenous infection has followed vaccination, circumcision and even trivial injuries to the cuticle, but the chief cause of tuberculosis following injury and shock must always be endogenous, that is, from within, the disease extending to an injured or weakened part owing to a lowering of body resistance. It is well known, for instance, that a tuberculous gland may bring about a metastatic infection after an injury to the gland. It becomes necessary to differentiate if trauma and shock have produced tuberculosis in a perfectly healthy but infected individual, or in one who was previously tuberculously diseased, but the process at the time was not active. Trauma and shock may be the necessary factors which aggravate a previously existing process or make an active one more active or one which has become inactive or quiescent again active, much depending if the organism or even individual organs have been weakened from intercurrent conditions previous to the injury, like overwork, grief, homesickness and other causes as have already been enumerated, all of which are important elements in the lowering of body resistance.

Into this category may also be placed all those vicious conditions which if indulged in over protracted periods are generally followed by a lowering of the body's vitality, especially long periods of dissipation and carousing, leading a vicious life, sexual and other excesses, irregular mode of living, abuse of the body, insufficient or unwholesome food, etc.

Tuberculous disease as it relates to trauma and shock may be divided into two classes, either into (a) **the class in which traumatism itself**, attended with little or no noticeable element of shock, is **the causative factor** or into (b) that class in which **shock appears to be the main cause.**

(A) In cases of tuberculosis in which trauma *per se* seems to be the exciting cause the process usually runs a rapidly fatal course. I wish to cite a few cases from the literature to which I wish to add one that only recently came under my observation. Ferdinand Weil M.M.W. 1910, No. 3, reports two cases, one of which during life was considered septic, the other typhoid. In both there was fever, rapid pulse and high temperature, with indefinite chest findings and in both an abortion was performed a week or so before the onset of the symptoms. Death about 5

weeks from the beginning of the disorder, postmortem confirmed the suspicion of miliary tuberculosis and in addition, in both instances the uterus was found to be the seat of a primary (?) tuberculous disease. Here undoubtedly, trauma to the uterus incident to the abortion brought about the entrance of tubercle bacilli directly into the lymph or blood stream resulting in disseminated tuberculous disease. Rose M.M.W. 1909, No. 38, reports a similar case, running a very rapid course, which during life was considered one of puerperal sepsis until the postmortem revealed its true nature. Marcus Posen; Monathschrift fur unfallheilkunde 1910 No. 10, reports a case of chronic encapsulated tuberculous gland in the axilla followed, after a severe contusion of the shoulder and injury to the gland in a man 43 years of age, by miliary tuberculosis and exitus in three weeks from date of injury, accompanied by meningitis tuberculosa. F. Parker Weber Brit. Med. Jour., 1910, reports a most interesting case. A young man in apparently perfect health, on shipboard, returning from Africa, received a severe blow on the testicle on April 1, 1897, and died 25 days after from miliary tuberculosis, and the postmortem showed distinct evidence of earlier lesions in the apices of both lungs, enlarged mediastinal lymph glands, evidence of an old pleurisy and a caseating mass in the testicle. The following case came to my attention a few years ago:

Dr. D., aged 54, died January 9th, 1918, of miliary tuberculosis. He was of healthy, sturdy Scotch stock with negative family history as to tuberculosis, had a family consisting of a wife and two daughters. About 10 years ago one of his children was taken ill with a most severe type of malignant scarlet and succumbed within a week of its onset. The shock incident to the death of the child brought on a severe nervous disturbance which was followed by universal Alopecia. In late years, and up to the fall of 1917 he was enjoying unusually good health having gained some in weight; however, during the summer he complained of an occasional attack of sore throat, a slight pharyngitis. On September 1, 1917, while removing his collar he noticed a slight swelling on the left side of the neck below the jaw near the posterior horn of the hyoid; this was diagnosed as an acute inflammation of the thyroid, an acute thyroiditis, and the swelling becoming somewhat painful and fluctuating, an incision was made September 6th, discharging about 15 cc of fluid pus. The gland continued discharging pus for some time when under gas an attempt was made to reach the bottom of the abscess so as to favor more rapid drainage and healing and after this procedure the pus soon ceased to flow and in its place a clear serum, simulating synovial fluid, kept flowing from the sinus and which greatly irritated the skin about the neck and shoulder. He now began showing symptoms of denutrition and this was attributed

to the constant loss of thyroid secretion, a disturbance in metabolism. Up to this time the physical examination of the lungs always gave negative findings but now the examination revealed a dull area, note high, bronchial breathing, whispering voice increased, posterior to the left of the spine from the first to the fifth dorsal vertebra and beyond the parasternal line, and suspecting the presence of enlarged bronchial lymph glands a stereo-roentgenographic picture was taken which showed a distinctly dense hilus shadow; in fact, the shadow was so dense that two careful and well-informed roentgenologists gave their opinion that it is more likely malignant than tuberculous. He now began to show a slight hacky, non-productive cough and after many attempts at securing a little sputum finally succeeded one morning in raising a little pelicle which on examination showed many tubercle bacilli and now an examination of the fluid secreting from the thyroid gland was made and it also showed the presence of the Koch's bacillus in large number. On December 5th, 1917, a second stereoscopic set of plates were taken showing unmistakably the presence of a miliary tuberculosis, death following within a month.

(B) Cases of pulmonary tuberculous disease in which shock alone seems to be the exciting cause. Reference has been made above that in surgical shock an abnormally low blood pressure is the most essential, single, phenomenon. It is well known that in pulmonary tuberculosis in either the active, inactive or arrested stage, low blood pressure is also a constant phenomenon; in fact, low blood pressure in an apparently healthy individual, with negative physical findings, is a most positive single sign of a present or future tuberculosis. From what has been stated we must acknowledge that shock causes a vasomotor dilatation, accompanied by damming back of the blood in the veins and capillaries, a slowing of the blood current, a venous stasis. We all are also familiar with the picture of the contracted chest in the young adult with its small upper aperture and its great tendency and frequency to tuberculous disease. We have here a similar phenomenon, insufficient aeration, lymph and blood stasis, with a strong tendency to metastatic disease. With a rapid or normal blood current, if bacilli should have found their way into the circulating fluid, they do not find easy lodgement, besides the fast flow destroys many, whereas a slow current presents all the elements favorable to deposition and growth of the tubercle bacillus, hence in shock the one symptom of low blood pressure, with stasis and vasomotor paralysis are just the factors which we all recognize as favoring the development of tuberculous disease. If the blood pressure in the tuberculous adult, with either an active, inactive, quiescent or arrested process is usually low and

if low blood pressure is an important phenomenon accompanying shock, then individuals who are on the borderline, in whom the physical signs are perhaps negative, perhaps slight prodromal symptoms or slight toxidity from slight tuberculin elimination and absorption, showing that somewhere in the economy a slight activity is taking place, pulse slightly accelerated, a slight variation in temperature, blood pressure a trifle low, if in such an individual, the blood pressure is still lowered by trauma and deep shock, which lower vitality and resistance, than the organ which already shows a lowering is usually the one which shows the disturbance and as at puberty and for some years beyond the lungs are most vulnerable, the other organs at this time in life have acquired a certain degree of immunity, it is now the pulmonary circuit which shows the effect of this induced shock.

How shock may bring about tuberculous disease may be well illustrated by the following case histories:

Case 1 and 2. Family consisted of Mr. D., aged 73; Mrs. D., aged 65, and four children, two boys and two girls; negative family history for tuberculosis. Miss A. L. D., age 27; occupation: bookkeeper; usual weight, 146 pounds; always enjoyed fairly good health and, with the exception of an attack of chicken pox at the age of 2, had no other children's disease. In July, 1911, while on a vacation, she met with a very serious accident. While riding in a street car at Niagara Falls, the car coming to a sudden stop she was thrown violently forward, her face and mouth coming forcibly in contact with the back of the seat in front. Since childhood, she had a faulty formation of the teeth which at the time were being corrected, the teeth being held in proper position by gold wire. In striking on her mouth all these teeth became dislodged, this was followed by some hemorrhage. She showed much excitement and nervousness at the time, reached home about two weeks after the injury and at once began complaining of frequent headaches, and at times displayed a most violent and irritable temper. She returned to her work in the fall of 1911 and in January, 1912, began to complain of a constant tired feeling (Prodromal Symptom). A slight cough appeared in the spring and in August, 1912, she discontinued her work, her symptoms now becoming more pronounced, a diagnosis of pulmonary tuberculosis was made and gradually progressing to the terminal stage, died October 1, 1913. During all this time the expectorations were very scanty, little cough, and towards the close she developed a painful pleurisy. Close confinement to her home, with close application and long hours at office work, perhaps illy-ventilated, and an attended injury and shock in a person of sanguine or nervous temperament brought about a reactivation of a probable quiescent or healed lesion, or by lowering of vitality and resistance activated a latent process.

Early in November, 1913, I was consulted by the sister of the dead girl and there in taking the family history I first learned of the sad fate and early demise of this girl. The following is a short history of Miss A. D.,

age 21, sister of the dead girl; occupation, bookkeeper. Rheumatism at the age of 14; asthma in 1907, continuing for 4 years; previous to that an Eczema lasting for 5 years; pneumonia, 1905; pleurisy 3 years ago. Examination, November 19, 1913; pulse 92, temperature 99.8, weight 133 pounds. Physical examination: evidence of an old pleurisy left lung, drooping of the left shoulder, right upper lobe anteriorly note high, voice conduction increased, inspiratory musical rales, interrupted breathing. Posterior muscle spasm. Krönig right 5 cm, left 5.5 cm; re-examination, March 18, 1914: pulse 74, temperature 98.8, weight 141 pounds. From these findings and the family history I advised her to be under constant observation and care. Now seven years after she is in most excellent health, living under strictly dietetic-hygienic rules since the death of her sister. The brothers though advised of the gravity, refused positively to be examined until in the summer of 1917 I was called to the home of Mr. D. to examine one of the sons, who at the time was suffering from pulmonary tuberculosis, second stage. He was sent to the Municipal Tuberculosis Sanatorium, where he remained but a short time, returning home; died, November 22nd, 1917, aged 29.

History: Whooping cough at the age of 7, no other children's diseases; occupation, R. R. clerk; usual weight, 168 pounds. In October, 1911, and at the age of 23 he met with a most serious accident. The Steamer Pere Marquette was foundered in Lake Michigan in a storm and out of a crew of 38 men only 5 were rescued, he being one of the five. He was in the cold water for nearly one hour before help arrived. One of the four men who were rescued with him shortly after became violently insane and is now confined in an asylum; this seemed constantly to pray upon his mind. In January, 1914, he began complaining of being tired, languid, even lazy. He would accept a position, fill it but a short time, then quit, always complaining of that tired feeling (Prodromal symptom). His family would remonstrate, but he appeared indifferent, moping and sitting about the house. Habits, good. In January, 1917, he began to show definite symptoms of active tuberculous disease, took to bed in June, and exitus letalis in November, 1917. His mother has since informed me that although he was an active and ambitious boy before, that ever since the accident he was constantly complaining of a tired feeling and that he was much indulged, because of this condition. Since the death of this boy, the other lad, now aged 28, has also come under my observation, and though his findings are negative, owing to his family history, it is advisable to keep him under observation.

Case 3. Mr. H. J., age 22; born in Chicago; vocation, accountant; usual weight, 158 pounds; first came under my care December 3rd, 1917. Chest examination warranted a diagnosis of chronic pulmonary tuberculosis, second stage. Pulse 120, temperature 99, weight 136½, slight clubbing of the fingers; sputum, 2 to 4 tubercle bacilli in each field. History: Scarlet fever at the age of three, followed by otitis media, this for more than 17 years continued, off and on, to discharge pus. Soon after puberty he began leading an irregular life and at the age of 17 was a confirmed cigarette smoker, consuming from 15 to 20 cigarettes daily, remaining often out of doors the greater part of the night, returning home early in the morning

and, without rest or breakfast, go to work, and during all this time the ear trouble gave him much concern. At the age of 19, in 1914, he consulted a physician who advised the removal of the tonsils as a possible means of curing the chronic ear difficulty. In May, 1915, under local anesthesia, in the doctor's office he had his tonsils removed. This was followed by a most severe hemorrhage, lasting for hours. He complains of a tired feeling since that time, and since the operation, 5 years ago, he has worked in all but 9 months. The ear still discharged after the removal of the tonsils and the physician suggested an autogenous vaccine. This was prepared and for more than 3 months he received 2 to 3 intracutaneous injections every week which so irritated him that he positively refused to continue with the treatment. Was the loss of blood following the removal of the tonsils a contributing factor to his tuberculous disease? It is well known that every severe injury to the body, particularly if accompanied by loss of blood, will reduce body resistance and often from an inactive to an active or from a latent to a manifest disorder is early noticeable. This patient died December 4th, 1921.

Let us revert for a few moments to the records of the 257 tuberculous patients who were sent in 1915 from the Central Free Tuberculosis Dispensary to the Municipal Tuberculosis Sanitarium, 77 of which gave a history of having had an operation performed within a year or so previous to the onset of the tuberculous disease. Have these operations, on the 77 patients, been performed at a time in life when the patients' vitality was at a low ebb or possibly at its lowest point? The greater number were in a decade, covering the time from the 14th to about the 24th year, the operations consisting mainly of appendectomies, tonsillectomies, laparotomies, turbinectomies, removal of enlarged glands, etc.

Immediately before, at puberty and for some years beyond when the physiological balance in the young individual is greatly disturbed by new or dominating, emotional and sensual impulses, when there is greatest danger of lowering vitality, would it not be advisable to delay all operations that are not necessarily life saving until this equilibrium has again been fully restored, or if it is deemed necessary to operate to do it in the younger years before the disturbance of this balance? An individual who presents a picture of, or perhaps some of the physical signs pointing slightly to a suspicious tuberculous disorder, even if apparently healthy, and if in such a person we find a blood pressure not proportional to his age, in whom it would be most advisable to minister to the upbuilding of his body energy and resistance, and with it increase his blood pressure, would it not become

most important that we guard against all such agencies which have the slightest tendency to lower this pressure?

In preparing an individual for operation, we find that generally a careful examination of the heart and its sounds is made, a thorough urinalysis and a blood examination, the lungs are usually very hastily examined, but we seldom see even a casual or hurried blood pressure observation made. With beginning of puberty the greatest pulmonary tuberculosis morbidity and mortality begins. This is usually in the middle of the second decade of life and in the middle of the third a perceptible decrease in the tuberculosis mortality is beginning to manifest itself and at this time in adult life the period of greatest resistance is in evidence, and operation better borne, eliminating in a measure, at least, the element of shock. As operations[2] play but a part in the production of shock, it becomes most important, particularly at the tender years towards and after puberty, to guard against all such conditions which may lower vitality and in that manner bring about an active tuberculous process from a quiescent or latent one and fear and fright, automobile and railroad accidents, grief and homesickness play as important a role in producing shock as do operations. We have learned that all physical and mental disturbances are factors in producing shock, hence avoid, particularly in young adults all such influences which may induce shock, which lower blood pressure, temperature and pulse, which lower vitality, lessen resistance, especially at a period, at puberty, when the balance is such that a little more in one direction or a little less in another, may lead either to a state of perfect health in one way or to a steady progressive with ultimate death in another, from pulmonary tuberculosis, the white plague, the great disorder of the human race, the one in which we all are so vitally interested.

[2]An acute or perhaps latent infection existing at the time of the operation is responsible in a number of operative cases for a subsequent pulmonary tuberculosis, hence only to operate when the surgeon has the full assurance that there is no latent or acute tuberculous infection.

PART THREE

LABORATORY DIAGNOSIS

Laboratory Diagnosis in Tuberculosis

Microscopical. Chemical. Biological, Etc.

This comprises three chapters. First: a general consideration of the sputum, both the normal and that from the tuberculous subject and how to collect, prepare and stain it for microscopic examination. Attention is also given to the lymphocytic picture, so noticeable in the sputum of the tuberculous, as well as to the albumin reaction. Second: the hematology, here the blood as seen in tuberculosis is considered. The hemoglobin, the leucocytes and the erythrocytes of the normal as compared with the blood of the tuberculous subject; Arneth's blood picture and its significance; the various theories and the demonstration of tubercle bacilli circulating in the blood stream. Under serology the complement fixation test only is considered, this somewhat in detail. In the last chapter, the various methods for staining and detecting the tubercle bacillus in the different body fluids, in excretions, secretions and in the excrements are considered. This consists in a microscopic examination of the urine, milk, bile, feces, pleural exudates, spinal and peritoneal fluids, etc. (7) (8) (16) (37).

CHAPTER 36

THE SPUTUM IN TUBERCULOSIS

General Consideration. Secretions which are expelled from the mouth during the act of coughing bear evidence of a catarrhal condition along the respiratory tract. These secretions may be more or less fluid, may be very abundant and, as in bronchiectasis, may be expelled at certain intervals accompanied by much or little odor. The sputum from tuberculous patients is generally odorless or, at most, of a faint mouldy character, most frequently colorless, white or yellowish-white; it may at times be tinged with green, the evidence of the presence of large number of pus cells, and may also be slightly blood tinged or streaked with blood, though usually it is not bloody. It is most freely expelled in the early morning hours or on arising and becomes less as the day wears on and then occasionally there is an entire absence of all secretions. This does not, however, exclude nor disprove the existence of respiratory disease.

In ordinary as well as in tuberculous sputum various morphological elements are always present, the most abundant of which are: (a) Epithelial cells, plain pavement epithelium, chiefly from the buccal cavity; these cells are readily recognized under the microscope as flat, polygonal bodies with irregular outlines, and by their size and the large nucleus. (b) Pus cells,—these are variable in number, usually in proportion to an inflammatory process; they may, however, be so abundant so as to form the chief element. Erythrocytes may also be present in greater or lesser number depending upon whether much or little blood is exhibited. (c) In the exudative form of pulmonary tuberculosis with much tissue destruction and cavity formation, elastic fibres are usually present. These fibres appear either as isolated, detached filaments or collected in bundles, straight, coiled, or twisted. (d) Besides these elements, mucous corpuscles, fatty cells and fatty crystals, cell nuclei, granular debris, etc., occasionally bronchial casts, lung stones and fungous growths are demonstrable. (e) When a specimen of sputum has been properly prepared, stained and is examined microscopically, the

tubercle bacillus, if present, can be readily recognized. (f) The expectorated sputum is often contaminated with coal dust, which gives it proportionately a distinct blackish appearance. This is also often observed in perfectly normal sputum. (g) Biliary pigment in the presence of icterus may frequently be found in the sputum, as well as varying amounts of coloring matter derived from blood. (h) Another amorphous element of sputum is mucus, a secretion from the bronchial mucosa which gives sputum its viscid character. Albumin is occasionally present and presages an inflammatory condition of the bronchioles, alveolar ducts, or the air cells. The fluid portion consists chiefly of water and the more abundant the expectoration the more fluid or watery it is.

Sputum is generally classified as to color and appearance into (1) **mucous.** This signifies a watery substance expectorated by perfectly healthy people, or perhaps more so in those suffering from bronchial catarrh. (2) **Muco-purulent.** This consists of both mucus and pus cells, the latter separating freely from the mucoid or watery portion as a sediment when the sputum is placed into a bottle and set aside for a short time. This form of sputum is frequently found in simple bronchial catarrh but most pronounced in pulmonary tuberculosis. (3) **Purulent** sputum; **pus sputum.** This form of sputum, generally homogeneous in character, consists almost entirely of pus, is derived from pulmonary cavities due to the breaking down of tissue, usually a concomitant in the rapidly progressing exudative type of pulmonary tuberculous disease. This, at times, may consist of pure pus, if pus in the pleural cavity has perforated the pulmonary structure and is discharging through a bronchus (empyema). (4) **Bloody or sanguinous sputum.** Sputum tinged or streaked with blood is usually from the mouth, teeth, throat or nasopharynx, etc., and may also be tinged after a gastric or oesophegeal hemorrhage. If the sputum is mixed with much blood and is coughed up, it generally comes from a ruptured vessel in the lung tissue and is then known as a pulmonary hemorrhage.

The Sputum in Pulmonary Tuberculosis. In acute miliary tuberculosis the sputum does not differ either in appearance, color or viscosity from that expectorated in ordinary catarrh of the bronchial tubes. In the proliferative type of the disease with but little or no destruction of tissue the expectorated material generally consists of the secretions of the bronchial tubes; con-

sequently, in appearance at least, it is not distinguishable from that of catarrhal disease; by microscopic examination, however, the presence of the tubercle bacillus may be demonstrable and albumin by chemical test, proven.

The bronchial secretions at the beginning of the disorder are usually very scanty, and, as time goes on, the intensity of the bronchial catarrh increase. Simultaneously, degenerative changes take place in the bronchioles and the air cells and the resultant material of these changes now become mixed with the bronchial secretions and form part of the expectorated sputum. In proportion as the products of these degenerative changes are much or little, the expectorated material, the sputum, assumes the appearance which to the eye is usually designated as "Tuberculous Sputum." This becomes practically more and more noticeable in the exudative type of the disease or what is commonly known as the broncho-pneumonic form. Here generally cavity formation is the clinical picture, more or less rapid breaking down of tissue, with high fever, rapid pulse, much cough, loss in weight and appetite.

The sputum becomes muco-purulent in proportion to the severity of the tuberculous process. In the cirrhotic or fibroid type of the disease the sputum again changes. assuming again the appearance of that which we usually see in ordinary or bronchial catarrh. Here, at times, there may be an entire absence of sputum or the sputum may accumulate during the night and be freely, perhaps copiously, expectorated in the morning. With bronchiectatic changes the sputum may accumulate in the dilated bronchi and, then, usually with change of position, large quantities of sputum are coughed up; this may be accompanied by little or much odor. We see from all this that sputum in pulmonary tuberculosis is subject to great changes in fluidity, consistency, viscosity, and from absence of to more or less watery, to muco-purulent, even to purulent or bloody, and from odorless to more or less mouldy, even to fecal. With the formation of cavities the sputum often changes, assuming a slightly greenish tinge and becoming more ropy, tenacious, and mixed with air bubbles, and here the muco-purulent material from the vomicae does not sink to the bottom of the vessel into which it is expectorated but remains suspended by means of the incorporated air. This sputum, under the microscope, shows many pus cells, staphylococci, streptococci, some diplococci, epi-

thelial cells, elastic fibres, debris, coal dust, etc., interspersed throughout with few or many tubercle bacilli. In the sputum of the tuberculous, proteolytic, amylolytic and lipolytic ferments can be demonstrated.

How and When to Collect the Sputum. It is evident that the sputum of the tuberculous individual is subject to great variations, hence we must aim at securing it at a time when the anatomical elements promise to be most positively present. The tuberculous person or the suspected is asked to secure the sputum in the early morning. The patient should have a clean, perhaps sterile bottle, cup, or container within easy reach, and when he has a coughing spell should raise the material from deep down the bronchial tubes and spit it into the vessel at hand. If the sputum is to be examined for the tubercle bacillus, the early morning or first sputum is desired. In collecting sputum for examination, it often becomes necessary to teach the patient how to expectorate. Very frequently the sputum secured is that from the buccal cavity, or perhaps, from the naso-pharynx, as the patient simply spits or hawks and what he brings out is put into a suitable container and sent to the doctor's office or to the laboratory. Here the patient must be told that the material which he coughs up, brings up from the bronchial tubes, and not that which he spits or hawks up, is wanted. He should be told not to suppress the cough, but to favor expectoration and that what is wanted is not saliva from the mouth, but secretion and mucus from the deeper portions of his lungs. It frequently happens that the sputum expectorated during the day may be free from tubercle bacilli or they may be so few as to escape detection, and frequently in a beginning case, or one in whom we suspect tuberculous disease, the bronchial secretions are very scanty or perhaps none at all. A little maneuver must then be resorted to so as to aid us in securing an amount sufficient for examination. The giving of 10 to 20 grains of potassium iodide in a little water in the evening may often produce in the morning most satisfactory results. To tuberculous individuals who cough quite frequently but do not raise sputum, the so-called dry cough, ¼ grain of morphine should be given at bed time; this has a tendency to quiet the patient and his cough as well, and on awaking in the morning he usually expectorates quite freely with the first fit of coughing.

The sputum when collected in a suitable container should at

once, or as soon as possible, be sent to the laboratory for examination, particularly so during the warm or summer months; during the winter or in cold weather it is not necessary to submit the sputum for testing at once as an interval of a few days does not vitiate a microscopic examination. Should it be necessary to send the sputum to some distant laboratory, the adding of a few drops of carbolic acid or a little formaldehyde is desirable, just sufficient to inhibit putrefactive changes while in transit. To a small amount of sputum, about a teaspoonful, in a wide mouth bottle add 2 to 5 drops of carbolic acid, cork securely and label, giving the name of the patient, time of collection and date, wrap in suitable material or make a package which will allow safe transit.

All sputum not wanted for laboratory examination should be received in paper napkins or clean clothes and these deposited in suitable paper bags and destroyed in a furnace—this should be a daily routine duty. Especially constructed sputum cups, made from various materials, chiefly heavy paper, are now much in use and are most appropriate for the collection of sputum. These cups are emptied every day or oftener and the contents consigned to direct fire or to specially prepared crematories. These sputum cups, or any sputum container, must always be closed after the sputum has been deposited so as not to spread the infection, as through flies. Cuspidors, so frequently seen at the bedside of home patients, should be strictly tabooed; if used at all, they should be one-half filled with water to which a teaspoonful of pure carbolic acid has been added.

To collect the sputum from children suffering from pulmonary tuberculosis is often fraught with great difficulty. Many children, while they do cough, do not expectorate, but seem to swallow the sputum. It is often very necessary to make a microscopic examination of the sputum but most difficult to secure a sample. Asking the child to cough and spit out is of no use, as it simply will not do so. Here, occasionally, more drastic measures must be resorted to. Place the child on a high stool or chair or preferably in the mother's lap, have an attendant hold the child's hands and feet, then place the head against your left side and with the left hand hold the child's head in a firm position; now ask the child to open its mouth, then place the thumb of the hand, which is holding the child's head, firmly against the cheek, and press the cheek well in between the upper and

lower jaw. With the index finger of the right hand covered with sterile or clean gauze, in the child's throat, move it about as if exploring the larynx and during the gagging which usually follows quite an amount of sputum can be collected for examination. The technic is quite similar to that for palpating a child's posterior nares for adenoids, differing only in that the method is here reversed, the index finger pointing downwards instead of upwards.

Examination of the Sputum. Microscopical and Chemical.

(1) **Microscopical:** (a) **The tubercle bacillus in the sputum. Preparing the sputum for microscopic examination.** If the sputum is fairly fluid, homogeneous, and easily miscible, then shaking well in a closed container may be sufficient. Should it, however, be thick or tenacious, then the adding of equal parts of distilled water and shaking may suffice. In most of the laboratories in the Boston hospitals, the sputum submitted for examination is immediately shaken with equal parts of a 2% carbolic acid solution till a homogeneous mixture is obtained. It is then ready for spreading on the glass slide. This is known as the Boston Method. Some laboratory technicians select with a platinum loop or sterile applicator a small nodule or cheesy mass, place it upon the slide, and, by moderate pressure, rub it into a uniform pulp and spread it evenly. Others add a small amount of formaldehyde and mix it intimately with the sputum. If equal parts of either distilled water or carbolic acid solution and sputum have been mixed, the number of bacilli observed in a stained field should be multiplied by two.

When the sputum is properly prepared then a few loopfuls are transferred to a glass slide, evenly spread, allowed to dry without heat, and passed through a bunsen or alcohol flame three times, after which it is ready for staining. The staining material in general use is a carbol-fuchsin solution. This dye stains the organisms a deep red, the bacillus holding this color with great tenacity. A few drops or sufficient to cover the sputum are placed upon such a prepared slide and, with a pair of forceps, the slide is held over the flame till the stain steams slightly. This is repeated several times, adding a little more stain. It is then washed in tap water and destained or decolorized. Tubercle bacilli do not stain readily but if once stained they do not readily destain. For destaining, usually a 25% solution of either nitric or sulphuric

acid is used. I much prefer destaining with Ebner's fluid, a solution of hydrochloric acid and salt in water and alcohol. Pour the fluid, drop by drop, upon the stained slide, held slightly slanting, until it flows away colorless, wash again in tap water, counter stain with methylene blue, wash, dry, and examine with a 1/12 oil immersion lens. Many other stains may be used. A Gram stain is also serviceable. A staining method reported to give a larger percentage of positive results, at least 25% more, affording a slide which is more pleasing and more restful to the eye is the following.

An Improved Staining Technic. A small amount of mucopurulent sputum is transferred to a glass slide and a drop of 5% solution of caustic soda added, and thoroughly mixed, using heat till a gelatinous mass is secured. This is now evenly spread, then placed into a drying oven, and when thoroughly dried, it is immersed in carbol-fuchsin solution warmed at 98.6° F. for 15 minutes, washed with equal parts of Esbach's fluid and water, decolorized with 25% nitric acid and, when faintly pink, washed in water, then in 60% alcohol, and again in water. Counterstain with malachite green, 1 to 20 (1 part saturated alcoholic solution and 19 parts distilled water), for 30 seconds, rinse, dry, and examine.

If in given sputum samples collected at various intervals from a suspected person, the tubercle bacillus, even after frequent examination, cannot be demonstrated, recourse should be had to some of the newer methods, which aim at a concentration of the sputum, assuming that the bacilli are but few in number. The Uhlenhuth or Antiformin method, the Ligroin method, according to Lange and Nitsche, and the Ellermann and Erlanderson method will all be found most dependable. These methods for the preparing of sputum are also advisable in those so-called closed cases in which we cannot demonstrate the tubercle bacillus after oft repeated examinations. In these cases the concentration of the sputum collected during the entire 24 hours will show very frequently that, after all, they were not closed cases, but that the bacilli were so few that they entirely escaped detection.

Much's Granular Stain. The granules left after the tubercle bacillus has undergone retrogressive changes are not stainable with the ordinary carbol-fuchsin solution and to detect the pres-

ence of these granules (and they are still virulent) in sputum or tuberculous material Much has devised a modified Gram stain, which is now known as the Much's granular stain. A slide is prepared in the ordinary way, then covered with Lugol's solution, heated over a flame until it begins to steam, and allowed to cool; this is repeated two or three times. It is then washed in tap water and dried between filter paper. Next, the specimen is similarly treated with gentian-violet solution and, after washing, is decolorized with 25% sulphuric acid solution, then alcohol or, preferably, at once with Ebner's fluid, it is then rinsed in distilled water, again dried between filter paper, immersed for about ten minutes in equal parts of acetone and alcohol, counter stained with methylene blue (1% solution), washed, dried, and examined.

There is neither a diagnostic nor a prognostic value in the number of bacilli demonstrable in a given sample of sputum. The number of bacilli found on a simple slide is wholly dependent upon chance. There may be very many bacilli in a given field and, then again, there may be very few in the next; hence, any quantitative scale devised or suggested is valueless as to the condition of the disorder or as to the prognosis. The scrutinizing of ten fields on a given slide, as first suggested by Ritter, counting the number in each, dividing by ten, and estimating the average number of bacilli in a single field in that manner is as dependable as any. Many laboratory diagnosticians make use of the Gaffky Scale for indicating the number of bacilli present and, from these findings, estimate the relative gravity of the pulmonary disease. All such scales are only arbitrary, expressing no true or definite condition as to amount of lung involved. The Gaffky Scale is as follows:

No. I—If, over a complete smear, only from 1 to 4 T. B. are found.
No. II—If, after examining many fields, a single bacillus is found.
No. III—One bacillus in every field (average).
No. IV—Two or three bacilli in every field (average).
No. V—Four to six bacilli in every field (average).
No. VI—Seven to twelve bacilli in every field (average).
No. VII—Many bacilli in every field.
No. VIII—Numerous bacilli.
No. IX—Very numerous.
No. X—Enormous numbers in every field.

Solutions used for staining and destaining tubercle bacilli:

Ebner's Fluid:

Acid Hydrochloric, C. P.	
Sodium Chloride, C. P., each	2.5
Distilled water	100 cc.
Alcohol	500 cc.

Gentian—Violet Stain:

Gentian—Violet	1 gram.
Phenol, pure	5 grams.
Alcohol	10 cc.
Water, sufficient to make	100 cc.

Esbach's Fluid:

Picric Acid	1 gram.
Citric Acid	2 grams.
Distilled water, sufficient to make	100 cc.

Ziehl-Neelsen—Carbol-Fuchsin Stain:

Fuchsin	1 gram.
Carbolic Acid, pure	5 grams.
Alcohol	10 cc.
Distilled water, sufficient to make	100 cc.

Lugol's Solution:

Iodine, pure	5 grams.
Iodide of Potassium	10 grams.
Water sufficient to make	100 cc.

Methylene Blue—A 1% Aqueous Solution.

(b) The Small Lymphocytes in Tuberculous Sputum.(82) Since the discovery of the tubercle bacillus, the examination of the sputum of the suspected tuberculous individual has rested mainly in a demonstration of either the presence or absence of these micro-organisms, and often the diagnosis has been based entirely on these findings, particularly in cases of doubtful physical signs. It is now well known that a sputum negative as to Koch's bacilli does not exclude pulmonary tuberculosis, but with the presence of albumin by chemical test, even in the absence of the tubercle bacillus, we may arrive at fairly definite conclusions. An anatomical element (other than the tubercle bacillus) is always present in samples of sputum obtained from tuberculous individuals; this is a preponderance of small lymphocytes. Such sputum is then always found to be albuminous.

Sputum lymphocytosis is evidence of a chronic pulmonary disorder for in the acute pulmonary disturbances, the polymorphonuclear type greatly predominates. As early as 1907, Wolff-Eisner directed our attention to an increase of these cellular bodies in the sputum of pulmonary tuberculosis. He states that

this noticeable increase in the small lymphocytes can be observed in every early case of pulmonary tuberculosis and that this total increase may number anywhere from 30 to 90%; that these small lymphocytes are always present, often long before the tubercle bacillus can be demonstrated, and that a sputum lymphocytosis is generally a forerunner of a tuberculous process.

(2) **Chemical Examination of the Sputum.**(78) The Albumin Test. The Albumin Content in the Sputum of the Tuberculous.

This is equally as important as the microscopical. If a microscopical examination determines the presence of the tubercle bacillus in the sputum then, in all probability, it is no longer a beginning case, the stage of incipiency has long passed, and the diagnosis is positively made. If, however, the signs and the clinical picture point to a suspicious pulmonary tuberculous disease and if microscopic examination does not prove the presence of the tubercle bacillus, then a chemical examination should be made to prove the presence or absence of albumin. A positive albumin content of the sputum, which is negative for bacilli, will usually give a positive intradermal tuberculin test.

The presence of albumin in the tuberculous sputum was first demonstrated by Rogers and Levy-Valenci in 1910. They reported on the sputum examination of 1200 cases of pulmonary disease representing the three stages, and only 17 gave a negative albumin reaction. They concluded from these observations that the sputum of the tuberculous generally is albuminous and that the absence of albumin always permits the elimination of tuberculous disease, but that the presence of albumin does not always allow a positive assumption that a suspected individual is actively tuberculous, because albumin is found in sputa of persons suffering from other lung affections. It is significant, however, that for a non-tuberculous person a negative albumin content is always a positive finding.

About the same time, similar observations were made by Lessieur who reported on 190 cases of pulmonary tuberculosis, in all of which he found an albumin positive sputum. He states that sputa containing the bacillus of Koch by direct microscopic examination will invariably give a positive albumin reaction; if, however, the sputa give a negative albumin test that microscopically or by any other method the tubercle bacillus can not be demonstrated. He further states that the albumin test is usually negative in all those affections which do not invade the

pulmonary parenchyma, but is always constant in the pneumonias, in pulmonary tuberculosis, and is often positive in cardiorenal disease. According to the observations of Rodriguez Alves, a positive albumin reaction is demonstrable in every phase of pulmonary tuberculous disease from the very incipient to the far advanced.

The sputa, collected from 108 clinical patients both suspected and ambulatory tuberculous, at the Rush Medical College Dispensary, were examined chemically for the presence or absence of albumin. The results confirmed the findings of the earlier investigators. A chemical examination of the sputûm of both the tuberculous and the suspected deserves of wider application. It is undoubtedly of great value in those very early cases when the clinical picture is not positive, where a positive sputum test may point to a positive tuberculous involvement and where the absence of an albumin reaction means negative as to tuberculosis.

The Great Importance of Properly Collecting the Sputum for Chemical Examination. The sputum should be collected, as stated above, early in the morning on arising. The patient should be asked to rinse the mouth with a few mouthfuls of warm water so as to free the mouth and throat from all adhering secretions. He should then cough up deep from the lungs and put the expectorated material into a clean, sterile, wide-mouthed bottle and close it securely with a well fitting cork. He must be specifically instructed not to collect the secretions from the mouth or throat, not the ordinary saliva, but the material which he coughs up and which comes from the lungs and bronchial tubes. A sputum submitted for testing should be examined at once or, at least, within six or eight hours, that is, before fermentative changes have taken place. In cold weather, a sputum one day old may still be suitable. No chemical antiseptic or preservative should be added to the so collected sputum.

Directions for Making the Albumin Test. As the technic is so simple, the results so satisfactory and the necessary apparatus so few, a chemical examination of the sputum should become the routine method in every case of either suspected or early pulmonary tuberculosis.

Put 5 cc of sputum into a glass cylinder of about 25 cc capacity (glass cylinder must be supplied with a well fitting ground glass stopper), add 5 cc of distilled water and about 10 drops of glacial acetic acid, replace the stopper, shake well, and set aside. Shake occasionally during the next 20 to 30 minutes, then proceed to filter. If the sputum is suitable for filtration it will have sep-

arated into three distinct layers, reminding one very much of an expressed stomach content ready for testing. Should the middle or watery portion of the sputum solution still be a little opaque, showing the presence of some remaining unprecipitated mucin, add a few drops more of the glacial acetic acid, again shake well, and set aside as before. The fluid portion now appearing clear, proceed to filter it through a plain, wetted filter, and test the clear filtrate for albumin in the usual way by either Heller's test or the heat test, either of which is very satisfactory; at my hands, however, the ferrocyanide test has proven most reliable and accurate. If, to an acidified albuminous solution of sputum in a test tube, a drop of 10 per cent ferrocyanide of potassium solution is added, an approximate idea of the amount of albumin can at once be gathered by observing the drop of ferrocyanide solution carrying down the precipitated albumin, and one can also notice the difference between a massive albumin content and one in which only a small amount of albumin is present by the manner in which the precipitated albumin falls to the bottom of the tube.

From personal observations, I have arrived at the following conclusions:

1. If the chemical examination of the sputum is albumin positive but tubercle bacilli negative, with physical findings obscure and indistinct, the intradermal injection of tuberculin will often change both to positive.

2. A positive albumin reaction and a positive tuberculin test are of far more importance diagnostically than the negative clinical examination; also, the negative value of the albumin and the tuberculin tests is of far greater, of more positive value than the negative clinical examination, because a negative clinical diagnosis may still be doubtful, may not be positively negative.

3. A single positive sputum finding can not be considered positively and diagnostically tuberculous, but a single negative albumin reaction can assuredly be considered as non-tuberculous.

4. A negative sputum for albumin is also generally microscopically negative for the tubercle bacilli; usually in the open or infectious cases both albumin and bacilli will be found in the sputum, while in the closed or non-infectious cases albumin minus the bacilli will be present.

5. The positive albumin reaction is a sure indication of an inflammatory invasion of the pulmonary parenchyma which may be tuberculous, pneumonic, or congestive, and the reaction is

always negative when the inflammatory process has not affected the substance of the lung, as in acute and chronic bronchitis and asthma; hence, the reaction is of most importance in those cases in which the pulmonary infection is not yet manifest and the differential diagnosis lies between tuberculosis and simple bronchial catarrh.

6. Albumin in the sputum is often the forerunner of the tubercle bacillus, and albumin may be found in the sputum many months before the bacilli make their presence known; hence, a positive sputum content, though bacilli negative, with but meager or very slight physical findings, favors a possible incipient or preincipient tuberculous diagnosis.

7. Like all clinical signs, it must be considered with all the other signs, both subjective and objective, to arrive at positive conclusions.

(3) **Combined Microscopical and Chemical Sputum Examination.**

In suspected or beginning pulmonary tuberculosis, the presence of a lymphocytic picture of the sputum under the microscope should stimulate a persistent search for the tubercle bacillus, and although they may be so few that they may escape detection entirely, even after oft repeated examinations, such lymphocytic sputum, if carefully observed at frequent intervals over long periods of time, will demonstrate sooner or later the presence of the tubercle bacillus. If we combine with these microscopic examinations a frequent chemical sputum test, we may arrive at a positive conclusion and clinch our diagnosis far in advance of being able show the presence of the tubercle bacillus.

"One hundred and ninety-nine samples of sputa collected at the Rush Medical College Dispensary were submitted to examination. First a careful microscopic search for the tubercle bacillus, then a study of the presenting microscopic sputum picture as to the preponderance of the small corpuscular element, and, lastly, a chemical examination for the presence or absence of albumin was made. The result of these observations may be briefly tabulated as follows. In fifty-three specimens tubercle bacilli were present—these samples were undoubtedly from individuals suffering from pulmonary tuberculosis; forty gave a distinct lymphocytosis and positive albumin content in variable amounts. Thirty-four were albumin positive and negative for both tubercle bacilli and lymphocytes. Seventeen were bacilli negative but showed a positive lymphocytosis of more than 50%. Again, seventeen were free from bacilli, lymphocytes, or albumin—these were samples from positively non-tuberculous persons. From the remaining thirty-eight sputa samples,

we have the following reports. The sputum was either too bloody for a reliable albumin test or it was distinctly leucocytic, or the lymphocytic picture was not sufficiently clear to be classed as positive, etc."

The parallelism between the lymphocytic sputum pictures on the one hand, and the positive albumin content of the expectorations on the other, in the absence of tubercle bacilli, was evident in so many of the above conducted chemical and microscopical examinations that on these findings alone a fairly positive diagnosis of pulmonary tuberculosis could be made, and, from the observation and study on these 199 samples of sputa secured from both tuberculous and non-tuberculous subjects, we are justified in permitting the following conclusions:

(1) A sputum lymphocytosis showing under the microscope 50% or more of small lymphocytes with a moderate amount of albumin by chemical test speaks very strongly for the presence of pulmonary tuberculosis.

(2) In beginning cases of pulmonary tuberculosis, even before positive physical signs are present, a sputum lymphocytosis can be demonstrated and with this an albumin content in varying amounts. The tubercle bacilli may be entirely absent or only occasionally a single bacillus found.

(3) The presence of lymphocytes in preponderant amount, with a positive albumin test, in a sputum sample is simply the forerunner or near the beginning of positive findings, and tubercle bacilli, if not already present in the expectorations, will soon be found.

(4) In sputum with a positive albumin reaction and giving a lymphocytic picture in the absence of the tubercle bacillus is confirmatory evidence that the sputum is from a person suffering from pulmonary tuberculosis.

A careful microscopic study of every sample of sputum under examination should first be made for either the presence or absence of the tubercle bacillus; if this should be negative, and a preponderance of the small lymphocytes and a positive albumin reaction be found, we are justified in assuming that the source of this expectoration is from a positively tuberculous individual.

CHAPTER 37

THE BLOOD IN TUBERCULOSIS

Hematology

General Consideration. Examination of the blood of a person with uncomplicated pulmonary tuberculosis reveals surprisingly little significant information. When secondary infection sets in a definite change becomes evident. (1) (45).

The Hemoglobin. In pulmonary tuberculosis, as a rule, the hemoglobin is normal or slightly reduced, even in the presence of pallor or marked cachexia; there are, however, individuals who show a marked reduction in hemoglobin, but these are exceptions. A hemoglobin below 75 is very uncommon, even during the active stage of the disease. We say that as a general rule the hemoglobin index in tuberculosis is good. This, however, does not always apply to the disease in children; where very frequently the hemoglobin is quite perceptibly reduced. In bone and joint tuberculosis in children, the hemoglobin is often considerably reduced, but usually not in children suffering from pulmonary disease.

The Erythrocytes. It is a significant fact that even in case of active tuberculous disease the red corpuscles are practically normal in number with a good color index. An initial rise in erythrocytes is occasionally seen in tuberculous patients after the first few hours of residence in a high altitude. This increase, following a residence in such altitudes is only a natural compensatory phenomenon—it is part of the process of maintaining the normal oxidation at such heights. In cavity formation with an accompanying fever, the red blood corpuscles are reduced, but where there is no fever, they are usually normal or perhaps slightly increased.

The Leucocytes. The white cell count varies but little from the normal, either in a count made at sea-level or at a high altitude. Usually the lymphocytes are somewhat increased, the count averaging, perhaps, somewhat higher at the high altitudes than at the lower levels; these appear to be increased in number

in the very early and in the favorable cases. The leucocyte count at sea-level is given by some as 4,000 to 5,000. This is regarded by many very competent observers as being too low and that 6,000 to 7,000 is nearer correct. The leucocytes usually remain normal in number and a leucocytosis is very exceptional. If, during a period of observation in a tuberculous individual in whom the leucocyte count has always been within normal limits, a sudden increase should be observed, it would suggest a beginning cavity formation. This results from the organisms of the secondary infection. So long as the leucocyte count is normal, cavity formation can be excluded, hence, a distinct leucocytosis in the course of active pulmonary tuberculous disease indicates the probability of cavity formation. It follows from this that a high leucocyte count is generally found in those cases with a bad prognosis.[1]

The Blood as a Whole in Pulmonary Tuberculosis. In comparatively few individuals suffering from pulmonary tuberculosis do we find both the red corpuscles and the hemoglobin diminished. The blood of the tuberculous person, however, as stated above, may show many changes if the products of mixed infection or if complications occur with the disease. The presence of organisms of secondary infection, septic conditions, prolonged diarrhoea, etc., all may have a more or less pernicious influence on the circulating blood. So long as the pulmonary disease remains purely tuberculous, very little change in either the number or the quality of the erythrocytes or the leucocytes is observed; the change only becomes apparent when secondary infection or complications occur.

According to Grawitz (89) the blood picture in the three stages of the disease varies greatly; if the diseased process in the lungs progresses, if there is much or little fever, or if cavities are present or absent. He notes the following changes. In the first stage of the disease, with infiltration and no signs of cavities, the erythrocytes are slightly decreased, the hemoglobin, about normal or perhaps slightly lessened, and the leucocytes are not changed. In the second stage, without fever, no complications and perhaps slight or beginning cavity formation, the erythrocytes are not changed, there is no change in the hemoglobin, but

[1](It should be noted in this connection that the injection of tuberculin is usually followed by a transitory increase of the leucocytes; this again changes in a few days, when the mononuclears again begin to increase slowly.)

the leucocytes are perceptibly increased. In the third stage, with hectic fever, the erythrocytes are greatly decreased, the hemoglobin, very much lessened or decreased, and the leucocytes, greatly increased. In female patients, in the first stage of the disease, there is frequently a decrease in the hemoglobin even in the presence of a normal leucocyte "count." In children, the clinical picture frequently seen during the second stage of the disease is that of oligemia. Such children appear pale, the skin is dry, the parts are much retracted, there seems to be a lessening of the whole blood quantity. In the third or cachectic stage, there is an increase in the leucocytes during the whole period of fever, due to the presence of pyogenic organisms. Some observers note that an improvement in the tuberculous condition is generally associated with an increase of the lymphocytes at the expense of the polymorphonuclears. If the patient grows worse then the polynuclears increase and the lymphocytes decrease.

In beginning tuberculous disease there is a gradual increase of the lymphocytes, and, as the disease progresses and assumes the form of mixed infection, the lymphocytes begin to decrease; with arrest of the disease there is again an increase of the lymphocytes. The polymorphonuclear leucocytes increase as the disease progresses, especially if toxic symptoms of mixed infection are manifest. Eosinophiles are increased if the disorder shows a healing tendency, decreased if the disease progresses. This picture keeps pace with the defensive powers of the organism.

Arneth's Blood Picture of the Polymorphonuclear Leucocytes in Tuberculosis.

According to Arneth there is a characteristic change in the blood of the active tuberculous which consists in an increase of the polymorphonuclear neutrophiles. The percentage of mononuclears (5%) to polynuclears (95%) in normal blood is about as 1 is to 20. In the blood of the tuberculous there is usually an increase in the mononuclears with a corresponding decrease of the polynuclears. This is dependent upon the assumption that the polynuclears are the chief elements in the warfare against the invaders and their toxins, that in this warfare many are destroyed; which liberate the contained antibodies, which now stimulate the production and formation of the mononuclears, the more youthful cells. (It should be mentioned that this theory is by no means generally accepted.) The neutrophiles, on account of their great preponderance, play an impor-

tant role. Arneth's theory is that the cells with the greater number of nuclei or of nuclear divisions play the leading part in combating infection and disease, and that the cells with the fewest number of nuclei are the immature or not yet fully developed cells, and that they take only a proportional part in the combat.

In health, there is a constant and definite proportion of the various cells to one another and in their number of nuclei; this, in disease, is greatly disturbed, the younger or more youthful cells predominating, and when such a change is observed in the blood picture, it indicates that the blood forming mechanism, the hematopoietic system, is greatly disturbed and can not meet the demands of the fight. It is thought from this that the polynuclears are the chief resisting cells of the body, being the first to show their activity in resisting the effect of infectious toxins, that the mononuclear are simply the reserve forces.

If a large number of blood examinations are made, selecting the blood from healthy individuals, a picture distinct and definite will be produced which is quite characteristic. One will readily observe that the nuclei in the polynuclear cells vary greatly as to the number of nuclear divisions, that the leucocytes containing three nuclei are most numerous, those with two nuclei are also fairly numerous, but less so than those with three, those with four nuclei less than those with two, that a fair proportion have a single nucleus, and but few have five. The average number, from a great many series of examinations, is as follows, per hundred cells:

In health	Number of nuclei	1,	2,	3,	4,	5.
	Number of cells	5,	35,	41,	17,	2.

From this count, Arneth suggested as an index the taking of the sum total of one and two dividing it by 3, 4 and 5, this gives 40/60 or ⅔. In the blood from tuberculous individuals, a considerable change in the polymorphonuclears from that of the healthy can be observed; the number of cells with a single nucleus increase considerably, those with two nuclei also increase, whereas those with three, four, or five perceptibly decrease, according to the following scheme:

In tuberculosis	Number of nuclei	1,	2,	3,	4,	5.
	Number of cells	16,	57,	26,	1,	0,
	with an index of 73/27 or approximately 7/3					

For these various indices, Arneth suggested the following arbitrary values:

Normal blood cells	with	one	and	two	nuclei		=40 to 55.
Tending to bad	"	"	"	"	"	"	=55 to 70.
Bad	"	"	"	"	"	"	=70 to 85.
Very bad	"	"	"	"	"	"	=85 and over.

We note here in the blood of the tuberculous a shifting of the nuclear cells from right to left,[2] that is, an increase of the mononuclears as well as the binuclears at the expense of the other polynuclear cells. This neutrophilic blood picture becomes of great prognostic value. If in the course of frequent blood examinations, the picture should show a gradual reversion from left to right, back to the normal, to the original type, fewer mononuclears and more polynuclears, then the prognosis would be much better, and inversely, with an increase of the mononuclears, the prognosis becomes proportionally grave, the fight against the disease being lost; hence, a rising Arneth index is regarded as indicative of a bad prognosis and a falling one as good and favorable.

The Arneth scale is discussed here because of the massive literature that has been written on the subject. It should be borne in mind that a large number of misconceptions have arisen from it and numerous conclusions have been read into it, many of which are quite foreign to the original intention of Arneth.

The Arneth scale is primarily a blood picture of the pathogenesis of general infection. He observes that when infection becomes ascendant there is an increase in the number of young forms of the neutrophiles containing but one granule, and a diminution of the older forms of the four and five lobed nuclear leucocytes.

The Tubercle Bacillus in the Blood Stream.(186)

There is no question in the study of the tubercle bacillus in which we find a greater difference of opinion than that concerning either the presence or absence of the bacillus in the blood stream. There is no doubt that in agonal or terminal cases the

[2]Arneth calls this a shifting of the blood picture to the left. Today we correlate this observation with injury to the hematopoietic system. It is part of the pathological alteration caused by the infection. Experimentally a similar blood picture can be produced by the administration of radium, X-ray, benzene and mustard gas, all of which have been shown to be leucotoxic agents. Today the leucocytes and lymphocytes of the blood are not regarded as important factors in resistance to tuberculosis although they may present evidence of the disease. Mallory and his pupils, also Klotz and his pupils have advanced the conception that the wandering endothelial phagocyte is the significant cellular element in resistance to tuberculosis and this view is rapidly gaining headway.

tubercle bacillus is frequently present in the blood current and can readily be demonstrated, but there exists much doubt if in beginning cases, or as some observers endeavor to show in the non-tuberculous, the bacillus is also present.

The bacillus was first isolated from the blood stream by Weichselbaum from three cases of miliary tuberculosis at autopsy, and by Meissel and others in acute miliary tuberculosis during life, but they were not able to demonstrate the bacillus in the blood from patients suffering from chronic tuberculosis. Later, other observers were able to show rod shaped organisms in the blood in the chronic form of the disease, Schnitter in 32%, Lippmann in 44%. In 1913, Brown offered a report at the meeting of the National Tuberculosis Association from observations made at the Trudeau Sanatorium. His observations were on 94 cases; bacilli were found in the blood in 4% of the incipient cases, in 19% of the moderately advanced and in 64% of the far advanced.

Kurashige of the Academy of Medicine, Osaka, Japan, examined the blood of his associates at the clinic, all apparently in perfect health. In 20 out of a total of 34 (59%) he asserts that he was able to demonstrate a bacillus in the blood. More surprising to him was the fact that some time later, in about 8 months, three developed a pleuritis, two had an initial hemorrhage, and in four others, animal experimentations gave tuberculous infection. From this, Kurashige concluded that very frequently in cases of pulmonary tuberculosis, the tubercle bacillus can be demonstrated in the blood stream, without resultant miliary disease. In a second report, he reaffirms his previous observation, emphasizing that the tubercle bacillus in the blood of the tuberculous person may be a constant phenomenon, and in a third communication, he states that the great source of tubercle bacilli in mother's milk is from the blood in which they always circulate.

Sturm states that in 40 to 50% of all tuberculous persons even in the early stage, the tubercle bacillus can be demonstrated but that their presence bears no relation to the severity of the disease; that 50% more are stainable by the Much modified Gram stain than by the Ziehl-Neelsen. Rumph (186) of Ebersteinburg, Baden Sanatorium, found four positive cases out of 18 examined (22.2%) and raised the questions whether these rods, found in the blood are morphologically and tinctorially changed, whether

the bacilli found in the blood are dead, weakened, dissociated, destroyed or devoid of a staining capsule, or whether the bacilli stain as bright and perfect when separated from the blood as they do in sputa. To answer these questions, his associate, Zeissler, examined the blood of 25 female patients who were in the incipient stage of the disease and found these so-called rods or tubercle bacilli in every instance namely, in 100%. He then examined the blood from seven supposedly healthy subjects and here also he found these same rods, again 100%. He next injected 35 animals with blood secured from a like number of tuberculous individuals in whose blood these same particular rods were found. Only three pigs became positively tuberculous, (8.5%), the others remained negative. The infection in one pig was from a case with a healed tuberculous lesion, the second pig, from a case of closed tuberculosis first stage, and the third, from a third stage case. All animals were killed thirty-one weeks after the injection.

Krause, reporting his findings on 100 patients in the first, second and third stage of the disease, states: Out of 55 in the first stage of the disease, the sputum was positive in only 10, the blood, negative in all; in fifteen second stage cases, with thirteen sputum positive and two negatives, the bacillus could be shown in the blood of five, all others negative; in thirty patients, far advanced, the sputum was positive in all, and the bacillus was found in the blood in 18 cases.

According to Liebermeister, the negative results obtained by animal experimentation for the purpose of detecting the presence of the tubercle bacillus in the blood depends upon various causes. The bacilli in the circulating fluid are comparatively few, and the greater part of the morphologically demonstrable bacilli are either weakened, damaged, or probably dead, besides the blood serum contains immunizing substances in relative stronger concentrations, the less diseased the condition of the individual is. In this manner, the animal is injected with a fluid which contains comparatively little virulent material suspended in a medium of great immunizing power. When injecting three or more guinea pigs with blood obtained from one and the same patient, at the same time, in equal amounts, and under precisely similar conditions, he observed that occasionally only one or, at the most, two pigs became tuberculously infected. This goes to prove that positive results point undoubtedly to live and virulent tubercle

bacilli in the blood, but it does not show that a negative result proves the absence of bacilli. The rapidity of the blood current may inhibit the growth of or even kill the bacilli, or the injected bacilli may not be capable of propagation, or, may perhaps, be lifeless or dead, or the immunizing substances in the blood may have inhibited their growth. Liebermeister succeeded in 40% of his animal experiments, in producing inoculable tuberculosis from blood of active tuberculous persons.

Technic for Preparing Blood for Microscopic Examination.

(A) The apparatus.

The necessary apparatus and solutions are the following:

(1) Centrifuging tube of 25 cc. This is known as the Zeissler

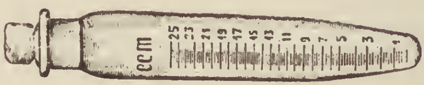

Fig. 60—Zeissler Tube. Used in demonstrating tubercle bacilli in the blood stream.

tube. It is graduated and supplied with a well fitting glass stopper (see cut above).

(2) A sterile needle or syringe for securing the blood.

(3) A laking solution. This consists of a 0.2% of neutral potassium oxalate solution. Dissolve 2 grams of neutral potassium oxalate in 1,000 cc pure, double distilled water, set aside in a closed container for 24 hours, filter rapidly through a thin, white, pleated, filter, after which it is ready for use.

(4) Pure concentrated antiformin.

(5) A quantity of 60% alcohol.

(6) Necessary pipettes, glass slides, flasks, etc., all scrupulously clean and free from dust.

The greatest care, accuracy, precision and cleanliness is required. All the necessary glassware should be washed in soda solution, then placed in sulphuric acid, rinsed in pure distilled water, then in absolute alcohol, and carefully put away in a dust proof compartment. The distilled water used for making the neutral potassium oxalate solution should be repeatedly distilled and of such purity as is now being used in lumbar puncture.

(B) The technic.

Having secured 5 cc of blood, put it at once into a Zeissler tube, add 25 cc of the potassium oxalate solution, shake repeatedly for about 15 minutes, and centrifuge, using a high speed,

electric machine. After centrifuging fully one-half hour, pour off the supernatant fluid, add 10 cc of pure distilled water to the sediment in the tube, and shake until a uniform emulsion is obtained; now add a drop of pure, concentrated antiformin, shake well, and set aside for a few minutes, after which add another drop and shake as before. This is to clarify the fluid; it usually requires but a few drops of the antiformin. After each drop added, shake, set aside and note if the solution is clear; this is generally found perfectly so in about one-half hour, after which the tube is filled with sufficient 60% alcohol and again centrifuged. Now pour off or with a large pipette remove all of the fluid portion, and in the sediment at the point of the tube these rods will be found. Remove one-half of this sediment with a hypodermic syringe and inject it at once into an experimental animal. Of the remaining half place equal parts on two glass slides, dry, fix, and stain one with the Ziehl-Neelsen the other with the Much granular stain. It becomes necessary to add a little albumin to fix the material on the slide.—J. R.

Serological and Immunological Considerations of the Blood.

Let us compare tuberculosis to some other infectious fevers with reference to the presence of the so-called immune bodies. Tetanus produces a toxin of extreme potency and two forms have been identified. A neurotoxin or tetanospasmin and a tetanolysin; none such exist in tuberculosis. Diphtheria produces a very potent toxin, a very small amount of which is fatal to the guinea pig; tuberculosis produces no such toxin. Rabies produces a toxin whose potency can easily be increased or diminished by certain cultural procedures; tuberculosis produces no such toxin. Typhoid fever produces specific antibodies—agglutinins which are specific against the cultures of typhoid bacilli in a dilution of 1 in 2,500; in tuberculosis the agglutinins occasionally reach a strength of 1 in 50, but these agglutinins are not always present in the blood of tuberculous individuals and frequently they occur in normal sera. There is not sufficient evidence on hand to regard them as being identified with immunity in tuberculosis if any immunity exists at all. In pneumonia and epidemic meningitis there are specific agglutinins against the various types of the respective organisms; in tuberculosis thus far no one has demonstrated a specific agglutinin against the human tubercle bacillus that will not in the same dilution agglutinate the bovine

bacillus. In certain infectious diseases one attack is generally regarded as conferring a permanent active immunity; in this group are typhoid fever, scarlet fever, small pox, measles, syphilis and others; this is not so in tuberculosis; all forms of contact with the disease render the organism only more susceptible to it. In certain other infectious diseases, a passive immunity can be conferred by the injection into the host of a suitable vaccine or its derivative products; in this group are smallpox, rabies, typhoid fever and to a slight extent diphtheria; no such immunity can be conferred in tuberculosis.

There yet remains to be demonstrated a single method of conferring passive immunity in tuberculosis in human beings, although every possible method has been tried. Dead cultures of all strains and all varieties have been tried as a basis of a protractive vaccine; so have living bacilli of both virulent and attenuated cultures, the bacilli and its products have been chemically split and divided in every conceivable way. There have been made water soluble extracts and alcohol soluble extracts, saline extracts and glycerine extracts, the bacilli have been used whole and macerated, triturated and inspissated and we are just as far today as our forefathers were in the days of Hippocrates so far as conferring passive immunity by any form of biological product which may be administrated to human beings. We are inclined to state quite definitely that there is no way of conferring passive immunity in tuberculosis; nor does one attack of the disease protect the organism against subsequent attacks; in fact the reverse is more likely to hold true. Every form of contact with this disease only renders the subject more susceptible to it.

From an immunological point of view we are inclined to follow others who state that there are no demonstrable immune bodies in tuberculosis; yet from a general biological point of view we are forced to conclude that there must be some form of biochemical protective mechanism or else we should all be dead of the disease. Just what this biochemical protective mechanism is we don't know but we feel that it is not antibody protection as we ordinarily understand immune phenomena. It is this biochemical protective mechanism or portions of it that we demonstrate by the indirect means of complement fixation.

Complement Fixation in Tuberculosis.

General Considerations. The discovery of the tubercle bacillus by Robert Koch in 1882 added a link to the scientific chain of

evidence which placed the pathological diagnosis of tuberculosis upon a practically infallible foundation. If the tubercle bacillus was found in the diseased tissues it made the diagnosis of tuberculosis certain. Unfortunately, in the majority of cases, tuberculosis is an internal disease and only in the advanced or ulcerative stages does it yield products to the external world suitable for diagnosis. The almost general presence of the tubercle bacillus in man, as determined by postmortem examination, adds a complicating influence to a clear definition of the disease.

The clinician is principally concerned in the separation of tuberculous infection from tuberculous disease. He wants to know definitely whether his patient has active or inactive tuberculosis, whether the disease is progressing or not, if it has any influence on the individual's physical ability, and what the probable outcome will be in the patient, under observation. In short, he would like a functional, diagnostic, and prognostic analysis of his patient. In view of the fact that the tubercle bacillus is so difficultly accessible in the human body, the search for the causative organism has been augmented by innumerable attempts to ascertain its presence by other means, and to determine whether or not it is causing destruction of the body tissues.

With the advent of serology, all of its methods were tried in an attempt to gain this information. The tuberculin test, although valuable in young children, was soon discarded as a diagnostic or prognostic agent in adults. Likewise, the precipitin and agglutination tests in tuberculosis were short lived, while the opsonic index method never did gain favor on account of the difficulties in technic and interpretation. Of the immunological tests, the complement fixation test has been probably the most elaborately studied and has given the most encouraging results. In order to comprehend the technical details of this test a few words on the history of the discovery may be helpful.

Historical Data in Complement Fixation. The complement fixation test is a reaction belonging in the immunological class of antibody reactions. The earliest observations on antibodies might be stated to be those made by Sir John Hunter, the celebrated English physician, who noted that unheated blood, on standing, did not putrefy as readily as heated blood. This observation paved the way for numerous others which followed, subsequently but rather slowly, but which eventually led to the

discovery by Richard Pfeiffer in 1894 of the Pfeiffer phenomenon. He noted that in a guinea pig which had been injected subcutaneously a number of times with cholera vibrio, and subsequently was given an intraperitoneal injection of cholera vibrio, there resulted changes in the intraperitoneally injected organisms which did not occur in those organisms also injected intraperitoneally in an animal which had had no previous inoculation. Those changes consisted in a peculiar granular appearance of the cholera vibrio and a final destruction by lysis of their bodies.

Metschnikoff and Bordet in 1895 demonstrated that this lysis could occur in the test tube provided the organisms were mixed with the serum from an animal previously given several injections of cholera vibrio. Bordet in 1898 elaborated on this work and demonstrated that red corpuscles would likewise undergo lysis both in animals previously injected with the foreign red corpuscles and **in vitro** when the serum of these animals was mixed with the homologous erythrocytes. He also demonstrated that in the typical **in vitro** red corpuscle lysis or hemolysis three constituents were necessary: (1) the specific red blood corpuscles which had been injected into the animal for immunizing purposes; (2) the serum of an animal that had been injected with these red corpuscles and thus had become immunized; and (3) the constituent found in all normal serums previously discovered by Buchner and called the alexin of Buchner, or, in our present terminology, the complement. Here then, we have the production of a specific lytic substance to an antigen, the red corpuscles which can produce an easily visible reaction (hemolysis), and it is this reaction which really laid the foundation for the Bordet and Gengou phenomenon of complement fixation.

Bordet and Gengou simultaneously noted in 1900 that red blood corpuscles, bacteria, or any antigenic substances could be sensitized by placing them in an immune serum (produced by injecting these same antigenic substances into animals) which had been heated for thirty minutes at 56° C, leaving the substance—"sensibilitrice" as they called it—(or amboceptor) uninjured, as it is thermostabile, and adding fresh serum containing complement (the latter being thermolabile and easily destroyed by heating at 56° C. for thirty minutes). The reaction between these three substances was a definitely quantitative one and could therefore be used for determining the presence of any bacterial antibody or red cell antibody complex.

Antigens. According to Hektoen an antigen is any substance which when injected into an animal body causes the formation of specific antibodies. As typical examples of antigens, we have most of the pathogenic bacteria, pathogenic protozoa, many albuminous bodies (i. e., venoms, enzymes, etc.), and bland proteins, (i. e., egg white, édestin, etc.). The action of a specific antibody, while specific, is strictly quantitative, depending upon the amount of amboceptors. As proof of this there exists the typical Neisser-Wechsberg phenomenon of deviation of complement which must not be confused with complement fixation. In the complement fixation, we have the union of complement with one lytic complex (antigen and amboceptor) to avoid its binding by another similar complex (antigen and amboceptor), but differing in specificity only; while in complement deviation, an excess of amboceptors binds the complement and thus avoids the action of the complement upon the antigen. Neisser and Wechsberg in 1901 found that a small amount of immune serum renders normal serum more bactericidal, but a greater addition of immune serum robs it of most and sometimes of all of its bactericidal power **in vitro.**

Bearing in mind the elements necessary for the mechanism of the complement fixation test, and also the definitely quantitative relations essential between the antigen, the immune body or antibody (amboceptor) and complement, we can intelligibly discuss complement fixation.

The Wassermann Reaction.

If, for instance, we wish to diagnose a certain disease by determining the presence of its specific antibody as, for instance, that caused by the cholera vibrio, we can do this in an indirect manner by making use of the red blood corpuscle complex which is easily visible to the naked eye. Let us, for example, call the antigen for cholera vibrio C, the immune body produced in the animal which has been injected with this organism CI and the complement CO. If we mix C, the known, CI, the unknown, and CO in quantitative relation, incubating for a certain period of time to allow fixation or binding, we get a combination of C, CI, and CO which completely fixes CO and removes from it the ability to react with any other lytic complex. If, then, we add to this red blood corpuscles, (e. g., human corpuscles) in a definite amount which we will call R, and an immune serum produced in

the rabbit by injecting with human red blood corpuscles, (this serum having been heated at 56° C. for thirty minutes to destroy the native complement) we have all the essentials for the production of hemolysis except complement. It is easy to see how the presence or absence of complement, dependent on its being bound in the cholera system that is complete, or not being bound in the cholera system which is incomplete, will give us a means of determining the presence of the unknown cholera immune bodies or antibodies used in diagnosis by complement fixation. Now we can substitute in this system any other complex as, for instance, syphilitic antigen and the serum from a suspected syphilitic patient and, by means of the red blood corpuscle system, always keeping in mind the quantitative relations in this system, can determine whether or not we have the serum of a syphilitic patient.

The Wassermann reaction, as we know it today, is one of the most practical of all complement fixation tests, but it was not the earliest application of complement fixation. Wassermann and Brück first applied bacterial extracts as antigens to tuberculosis, but found the test for tuberculosis impractical. These investigations occurred at a period when active search was being made for the spirocheta pallida, which was discovered by Schaudinn and Hoffmann in 1905. It then occurred to Wassermann that the fixation of complement test could be applied to the diagnosis of syphilis. His first results were published in 1906 together with those of Neisser and Brück. A report was also made at that time of experiments in monkeys in which salt solution extracts of syphilitic tissues were used as antigens.

The Complement Fixation Test in Tuberculosis.

The first application of complement fixation in tuberculosis was made by Widal and LeSourd (107) in 1901. They obtained fixation of complement in certain cases of tuberculosis, using as antigens homogeneous suspensions of tubercle bacilli of the Arloing-Courmont strain. In 1903, Bordet and Gengou (126) demonstrated the presence of an antibody capable of uniting with tubercle bacilli and fixing complement in the sera of tuberculous animals. Wassermann and Brück,(118) in 1906, demonstrated the presence of an antibody to tuberculin as antigen in patients treated with tuberculin, and, as a result of this, developed the

idea of antituberculin being formed as a result of tuberculin treatment.

About the best study up to 1913 was that made by Elizabeth Fraser (156) who compared a large variety of antigens prepared from tubercle bacilli and concluded that those prepared from living human bacilli were most reliable for tests in human tuberculosis.

Dudgeon, Meek and Weir, (155) in 1914, also tested a large number of antigens and found the bacilli or their products the most suitable antigens. McIntosh, Fildes and Radcliffe,(159) after testing a large number of antigens, also concluded that the bacillary emulsion prepared from live bacilli was best.

Craig (129) employed as antigen an alcoholic extract of several strains of human tubercle bacilli grown on a special egg medium, the antigen being an extract of bacilli and medium. Since then, alcoholic extracts have innumerable times been proved impractical, and a recent paper by Lucke,(149) in 1916, corroborated these earlier findings.(108)

In 1916, Corper (108) described an autolysate antigen which was prepared by autolyzing tubercle bacilli in physiologic saline for ten days at incubator temperature. The supernatant, clear, yellow autolysate was found to contain antigenic properties equal to that of a bacillary emulsion prepared from the same cultures and to possess the advantage of having a wider range between its antigenic and anticomplementary unit, than the bacillary emulsion.

In 1917, Corper and Sweany (109) compared the findings with the autolysate and Miller's (151) bacillary antigens, and found very little difference between the number of positive results obtained by means of these two antigens. They concluded that it is absolutely impossible to differentiate active from inactive tuberculosis by means of this test, and that the main value of the test was for differential diagnostic purposes in conjunction with the other findings in the case.

While the foregoing studies were being carried on by other investigators, Petroff of Saranac Lake was busy devising new antigens in the hope of finding an ideal antigen which would prove reliable. Beginning with a culture of bacilli grown upon a potato medium which did not finally prove satisfactory, he prepared four new antigens (143) a potato filtrate prepared from the filtered extract of a potato broth culture medium upon which

tubercle bacilli had been grown, a sodium hydroxide extract of tubercle bacilli, a methyl alcohol extract of tubercle bacilli, and finally a hot, glycerol "extract." (120) He maintained that in order to get good results a variety of antigens should be used.

In 1918,(150) Brown and Petroff, using these antigens, found a positive complement fixation in 72 per cent of 478 patients on whom a diagnosis of tuberculosis was made; 51% of the incipient cases; 73% of the moderately advanced, and 81% of the far advanced. When activity was present, 81% gave positive reactions, and when absent, 61%, while 90%, in whom tubercle bacilli were found in the sputum, were positive. They concluded that patients with a negative reaction could be allowed to exercise more freely than those with a positive reaction. Besides the comparative studies carried out by Fraser, Corper and Sweany, Petroff, and others, the work of Miss Lange (158) at Johns Hopkins Hospital is worthy of mention.

During 1918, there appeared a significant contribution by Heiman (157) as a result of a study of the complement fixation test for tuberculosis on children. He used Petroff's and Miller's antigens on the serums from 50 children ranging in age from six months to twelve years. Sixteen of these were tuberculous, six were suspects, and twenty-eight were non-tuberculous. Six cases of pulmonary tuberculosis (two with miliary) were negative with both antigens; one case of tuberculous peritonitis was negative; in six cases of tuberculous meningitis, four were negative and two, positive; seven cases of pleural effusion, of which one was definitely tuberculous, were all negative. Among the twenty-eight cases with no tuberculosis, three gave a strong positive reaction, one a slight reaction, and one a suspicious reaction. Heiman concludes that the test in children was of no value, but believes that a further improvement in technic may eventually make it of some value.

In 1919, there appeared several significant articles on complement fixation in tuberculosis. Young and Givler,(93) who used three antigens (Corper's autolysate, Petroff's methyl alcohol, and Wilson's bacillary suspension (95)) comparatively in a study of tuberculous, normal, and syphilitic serums, concluded that the three antigens did not differ greatly in the percentage of positive findings in known cases of pulmonary tuberculosis, while clinically normal individuals reacted to the extent of 11% with all the antigens. A fairly high percentage of luetic serums also gave

positive tuberculosis tests with all three antigens in the absence of demonstrable tuberculosis. Paul A. Lewis,(154) who made an effort to apply absolute quantitative methods to the study of this reaction, found that the serums of certain tuberculous individuals gave strong positive reactions, while others entirely failed to give a reaction. The serums of certain persons to all intents and purposes normal gave strong reactions also. He concludes from his work that the numerical relations are such as to make it unsafe to apply the complement fixation reaction to the diagnosis of tuberculosis except as a matter of the most limited confirmatory interest.

Having thus reviewed the views upon complement fixation in tuberculosis of some of the most reliable and most efficient investigators in the tuberculosis field and, from their observations and conclusions, we can sift out the following important points regarding this test in tuberculosis.

1. In spite of the fact that there is a large variety of tuberculosis antigens, there is very little difference between them in so far as their reliability and efficiency is concerned, especially with regard to the positive results obtained by their use. The essential point of importance is that they must be prepared from tubercle bacilli (preferably polyvalent) or suitable products thereof.

2. The percentage of positive tests obtained with the serums in cases of tuberculosis increases with the advancement of the disease, although very far advanced or moribund cases are likely to give a comparatively lower percentage of positives than might be expected from their stage of the disease.

3. Active cases of tuberculosis give a higher percentage of positive reactions than inactive cases, but the difference is not sufficiently great to be of diagnostic value.

4. Sputum positive cases of pulmonary tuberculosis give a higher percentage of positive reactions than sputum negative cases, but here also, there is not sufficient difference to be of diagnostic value.

5. In the presence of syphilis, or in a case with a serum giving a positive Wassermann reaction, the tuberculosis complement fixation test, if positive, is of very dubious value because a high percentage of luetic serums give cross fixation with practically all the tuberculosis antigens.

6. It is advisable to use the complement fixation test for tuberculosis with reserve, and, in the terms of Lewis, to apply the test in diagnosis as a matter of the most limited confirmatory interest.

7. It is possible, however, that from the standpoint of prognosis, according to Brown and Petroff, the test is valuable in that repeated tests at definitely spaced intervals may help to indicate the cases that may be allowed to exercise more freely.

Technic of the Complement Fixation Test for Tuberculosis.

The technic for performing the complement fixation test for tuberculosis consists essentially of the technic employed in performing the Wassermann test, with the exception that the luetic antigen is substituted by one of the common tuberculosis antigens.

For the complete details of the preparation of the red corpuscle suspensions, immune serums, inactivations, etc., the student is referred to the texts written on the Wassermann reaction, or to good text books on clinical and laboratory diagnosis. H. J. C.

CHAPTER 38

THE URINE, MILK, FECES, BILE, PLEURAL EXUDATES, SPINAL AND PERITONEAL FLUIDS, ETC., IN TUBERCULOSIS.

Bacilli. (A) In the Urine; (B) In Mother's Milk; (C) In the Feces; (D) In the Bile; (E) In Exudates and Fluids, etc.

The Urochromogen Test; Lime Salts Elimination; Cytodiagnosis; Inoscopy, Etc.

Examination of the Urine in Tuberculosis.

(A) **Characteristics of Normal or Healthy Urine.** (39) Urine, the fluid eliminated by the kidneys, received temporarily in the bladder reservoir, in health, is voided at practically regular intervals. This fluid, consisting chiefly of water, is separated by the kidney filters, the glomeruli and the solids and coloring matters, consisting of various mineral compounds and retrogressive protein bases, are secreted by the epithelium of the renal tubules. The latter, the proteins of the urine, represent the continuous waste taking place in the economy, a physiological process; consequently by means of urinary examinations, we are in a position to estimate the daily output as compared with the intake, all within normal physiological limits. In disease all this undergoes very noticeable changes, serious disturbances in metabolism effecting the urinary secretions, hence a study of the urine in the diseased individual often gives us more accurate information than may be obtained by either the thermometer or the pulse. In health, the daily excretion of urine averages 1500 cc, or about 50 ounces, the solid constituents, inorganic matter and mineral salts contained, about 70 gramms or 2½ ounces. In 1000 parts of urine there are approximately 35 parts of solid to 965 of water. A 24 hour specimen of urine is usually of a straw or light wine color, specific gravity of about 1.020, usually acid in reaction, and when freshly voided has a peculiar, slightly aromatic odor, and is always microscopically clear.[1]

[1]To readily determine the total solids in the urine, multiply the last two figures of the specific gravity by 2.33. For instance, the specific gravity of the urine is 1.020, then multiply 2.33 x 20 = 46.50. This gives the total solids in 1000 cc, equal to 46.60 grams to the litre.

(B) **Characteristics of the Urine in Pulmonary Tuberculosis.** Here the urinary output is greatly influenced by such conditions as pyrexia, diarrhoeal disturbances, night sweats, copious expectorations, vomiting, hemorrhage, etc., in all of which the volume of urine may be greatly decreased; it may, however, even be augmented if during such disturbances the intake of fluid is greatly increased. In the diarrhoeas, often accompanying active pulmonary tuberculous disease, the kidney elimination may be lessened by one-half of the usual daily quantity; this is also seen if so-called hectic fever is present; again, in those instances of active disease when the ingestion of food is almost immediately followed by vomiting, the urinary flow also becomes greatly reduced. This is again observed in the rapidly progressing, in terminal cases, and in the miliary form of the disease. So long as the appetite is good and the ingestion of food ordinary in amount, requiring the usual intake of water then there is no perceptible lessening of the urinary elimination. The decrease only becomes apparent when the disease is running a rapid course, when exacerbations and remissions follow in rapid succession. It is then that we observe a variation in the amount, color, and the general appearance as well. Then again, as in the normal, a large intake of fluid, as in the exclusive milk diet, the volume of urine is greatly increased, the urine appearing almost water white.

In pulmonary tuberculous disease whether accompanied by renal disorder or not, the urine often cannot be differentiated in amount, color, reaction, specific gravity, etc., from that passed by a healthy person, but by both chemical and microscopic tests the difference becomes quite evident, and the presence of the tubercle bacillus, the albumin, the abnormal lime salts, the nature of the coloring matter all become interesting factors in the examination of the urine in tuberculosis.

Tubercle Bacilli in the Various Fluids and Solids of the Body.

(A) **Methods for Demonstrating Tubercle Bacilli in the Urine.**(94) As early as 1882, Damsch inoculated guinea pigs with the urine of suspected tuberculous individuals to establish a diagnosis of renal tuberculosis, and in 1883, Babes demonstrated tubercle bacilli in the urine of patients suffering from genito-urinary tuberculosis. The method then in use was the sedimentation, or as it was called, gravitation method and later, centrifugalization (187) was the method of selection. From the

sediment in the urine glass or in the centrifuging tube, smears were made, dried, fixed, and stained in the usual way. The gravity method is still the one most in general use, and the technic as outlined by Walker in 1904 is the one chiefly used. From an exhaustive study of renal tuberculosis, he was able to demonstrate the bacilli in every one of the 50 cases studied. Walker's (141) Method is as follows:

(1) The specimen is allowed to settle in a conical urine glass for twelve hours.

(2) The sediment from the conical glass is taken up by means of a pipette and two drops placed on clean, sterile glass slides.

(3) The slides are put on a frame above a Bunsen flame and slowly allowed to dry—avoid rapid heating.

(4) The slides are passed through the flame in the ordinary manner so as to fix the specimen.

(5) They are then placed in a 5% acid (HC1) alcohol solution and allowed to remain for five minutes. This dissolves the urinary salts.

(6) The slides are then washed in running tap water to remove all traces of acid and alcohol.

(7) The smears are now stained in the ordinary way with carbol-fuchsin and decolorized with Gabbett's Methylene blue.

(8) Wash in tap water, dry, and examine.

Other methods in use are, the Uhlenhuth, Antiformin, the Ellerman and Erlandsen, Pancreatin and Sodium Carbonate digestion, the Lange and Nitsche, Ligroin, the Kozlow, ether and acetone, the Petroff, Acetic and Tannic Acid methods, etc.

A modified method similar to Walker's which perhaps may promise more definite results is one in general use at The Rocky Glen Sanatorium. (191) The following instructions are given:

Acid Fast Bacilli in the Urine. Urine was collected from female patients only. The patients were instructed to urinate into three vessels, the first two were discarded as they were intended to act as a wash for the meatus and vulva. The third specimen was used for the test. The tests were made upon women only as there is less apt to be smegma bacilli about the meatus in women who are in an institution, than in men.

(1) The urine is centrifuged for one hour at a rapid speed.

(2) A small amount of the sediment is placed upon a clean glass slide. The slide is permitted to dry slowly over a bunsen flame.

(3) As the sediment dries, a very small amount of egg albumin is stirred into it. If the specimen contains albumin it will not be necessary to add egg albumin.

(4) When the specimen is perfectly dry, pass through the flame ten or twelve times.

(5) Dip the slide in a 5% acid alcohol (HCl) solution. This will dissolve some of the uric acid salts. Wash in tap water.

(6) Dry slide, and again pass through flame.

(7) Steam 10 minutes with a carbol-fuchsin solution.

(8) Wash slide thoroughly with distilled water.

(9) Decolorize slide for 1 minute in a 1% acid (HCl) alcohol solution.

(10) Wash off thoroughly with water.

(11) Counter stain with a methylene blue solution containing 25% sulphuric acid (Gabbet's stain). (This acts as a counter stain and also as a second decolorization to make sure that none but true acid fast bacilli hold the carbol-fuchsin stain. Gives more decolorization than used for sputum analysis).

(12) Wash, dry, and examine slide.

To secure urine from male tuberculous patients and to avoid contamination with the smegma bacillus, it is advisable to secure the urine by catheterization.

The acid fast bacilli when present in abundance will almost always occur in large groups surrounded by a peculiar capsule or halo. Only one or two such large groups may be found on an entire slide. If not present in abundance, they may be grouped together, and in most cases they are typical, absolutely, of the tubercle bacillus. In patients with negative sputum, mostly first stage cases with very little active chest findings, the organisms may appear somewhat shorter than the bacillus usually found in sputum, but when the urinary sediment is injected into guinea pigs, pure cultures of typical tubercle bacilli are reproduced.

Mark's observations cover 150 cases with 600 urinary examinations, 45% first stage, 20% second stage and 35% third stage cases. 40% of the cases showed acid fast bacilli in the urine at some time only, and in 60%, the bacilli were found in every test. These laboratory observations are of special interest. He found that incipient cases with negative sputum, ones that had few active findings, the most constant common symptom being "tired feeling," showed a larger percentage of acid fast bacilli in the urine than any other. Cases with positive sputum in the in-

cipient and moderately advanced classes seldom showed acid fast bacilli. His interpretation is that when bacilli are expectorated there is less tendency of their getting into the blood current, and that in cases in which the condition is active but no connection with a bronchus the organisms may gain entrance to the blood, being filtered out by the kidneys without any apparent kidney disturbance. Other observers have also described the tubercle bacillus present in the urine in the absence of positive renal disease.(115)

May not the kidney permeability, which we occasionally observe in children and young adults (which in the literature is described as orthostatic albuminuria) and which I have considered as a prodromal (78) symptom (see Chapter XI, "Symptomatology"), give after all a bacilli positive urine in which the bacteria may be demonstrated if we only search diligently and persistently? The urine test for albumin can be made by any of the well known methods—the Heller test, the Acetic Acid and the Ferrocyanide test, etc. This is fully given in the chapter on the Sputum in Tuberculosis, and as the tests there given are all applicable to urinary examinations, the reader is referred to its pages.

(B) **The Demonstration of Tubercle Bacilli in Mother's Milk.** Judging from the nature of pulmonary tuberculous disease it is presupposed that tubercle bacilli may be present in the milk of the pregnant tuberculous woman. Available and reliable statistics from the literature regarding the bacteriology of human milk, however, are very meagre and even what has been done by different observers reveals a lack of concerted conclusions. According to Holt, tubercle bacilli have been demonstrated by Rogers and Garnier in the milk of a woman with advanced pulmonary tuberculosis, but that ordinarily they are not present unless the mammary glands are tuberculously diseased, and Marfan, referring to this case and a case reported by Demme, states that these are the only two authentic cases of children being infected from the mother's milk, but he contends that nevertheless the tuberculous mother in the interest of both herself and offspring should not nurse her child. Bandelier and Roepke state, "The danger of infection from mother's milk is very small when compared with the much greater one, the natural, the most intimate relationship existing between the mother and child."

Wang and Coonley, (131) reported their observations on 28 tuberculous women after childbirth. These examinations were made twice each week securing from each patient from 3 to 5 cc of milk, testing the same not later than two hours after it

was withdrawn. From each specimen secured, separate smears were at once made, and the balance reduced with a weak antiformin solution. The milk was mixed with equal parts of a 5 to 10% antiformin solution, immediately placed into an incubator for one hour, then centrifuged at high speed for 10 minutes, the supernatant portion removed, the sediment in the tube thoroughly mixed with distilled water, and recentrifuged for 10 minutes. From this second sediment a few drops were placed on a glass slide, egg albumin added, dried, fixed, and stained in the usual way. The sediments secured from fifteen patients in this manner, after the smear preparations were injected into guinea pigs intraperitoneally; these were all negative. The milk secured in this series was from patients in the moderately or far advanced stage of the disease, ranging in age from 19 to 40. The total number of microscopic milk examinations made was 450, taken biweekly; all were negative but one, this from a pulmonary-orthopedic patient. The woman insisted upon nursing her child and now, 1½ years after, this child appears perfectly healthy and is well developed. From these observations they concluded that tubercle bacilli (living?) are infrequently found in breast milk of the tuberculous woman who has no mammary disease; that though the possibility of infection from this source is slight, it is best to interdict nursing, for both mother and child, as perhaps toxic substances, other than bacillary, in the milk, may have a most deleterious effect on the young organism.

In all probability, if the tuberculous process is not very extensive, perhaps only few tubercle bacilli are circulating in the blood current, then few or none will enter the milk from this source; perhaps those that may enter are dead, destroyed, weakened, or non-virulent, and the milk will not infect the nursing child. In advanced tuberculosis, when the milk glands themselves have become tuberculous, and there is a great possibility of their contents contaminating the milk, the danger to the child becomes enormous, hence to guard the child and in the interest of the mother, it becomes imperative that the mother must not, if tuberculously diseased, nurse her child.

In this connection the following observations made on animals may be apropos. The Royal English Commission on Human and Bovine Tuberculosis in its third report in 1909 makes this statement: "If milk from clinically tuberculous animals is injected into the experimental animal, the result will always give a positive reaction even if on postmortem the udder is found free from tuberculous disease; if, however, the milk from a cow in

which the only sign is the positive tuberculin test is injected into such a test animal, the result will always be negative; hence clinically tuberculous cows without disease of the udder may contaminate the milk with tubercle bacilli, but more often the milk is entirely free."

It should be mentioned here, that the examination of milk for tubercle bacilli, secured from milk producing animals, cows, goats, sheep, etc., does not differ from the method given by Wang and Coonley for testing the milk of the tuberculous mother.

(C) **Tubercle Bacilli in the Feces.**[2] Tubercle bacilli if found in the feces are generally of pulmonary origin. In active pulmonary tuberculous disease when the sputum is bacilli laden, many will find their way into the oesophagus, thence into the stomach, from there into the intestinal tract, and be passed out with the stool. As a rule the bacilli in this passage are not destroyed nor dissolved; they are found to be very resistant against the digestive ferments and are not influenced by decomposition processes. In primary intestinal tuberculosis, bacilli may frequently be passed with the stools. However, if the retroperitoneal or the mesenteric glands are tuberculously diseased and are subject to degenerative changes still the bacilli infrequently find their way from this source into the intestinal tract.

The character of the stool usually speaks for or against enteric disease. A stool containing much mucus, blood, and some pus, a thin discharge or diarrhoeal stool, is in all probability from a patient suffering from intestinal tuberculosis. v. Jaksch is of the opinion that a positive diagnosis of ulcerative, intestinal tuberculosis can only be made if the bacilli are constantly present in the stool, particularly if they are present in pure culture if in large groups and always if pus is present. If in the surface washings of a fully formed stool the tubercle bacillus can be demonstrated, it in all probability is from the intestinal walls, from the intestinal mucosa, and points strongly to intestinal tuberculosis. If, however, in a fully formed stool the bacilli are found in portions from the interior, these in all probability are from the ingested sputum and are of pulmonary origin. The giving of an opiate, therefore, to lessen peristalsis so as to temporarily check the bowels may insure a well-formed, solid stool,

[2]The smegma bacillus is said to be frequently found in the anal orifice and may become mixed with the stools; this should be borne in mind when making fecal examinations.

and here the examination of either surface or interior may point to the source of the bacilli.

A very practical method for examining feces for the presence of the tubercle bacillus, based upon the suggestion of Hamburger, is given by[3] Strassburger.(192) This method is based upon the fact that in order to remove bacteria from a fluid it is advisable to lower its specific gravity, particularly so if only few organisms are present and this lowering of the specific gravity is readily accomplished by the addition of alcohol. Equal parts of water and absolute alcohol lower the specific gravity to 0.8975 at 15° C. Accordingly, a small particle of fecal matter about the size of a pea is rubbed up well in a mortar with a small quantity of distilled water, centrifuged for a very short time to eliminate practically most of the coarser particles, the bacilli remaining in the supernatant fluid; this cloudy fluid is now poured off, mixed with an equal volume of 95% alcohol, and then well centrifuged, the fluid portion decanted or removed, and the sediment in the tube fixed and stained in the usual way. To lower the specific gravity of a fluid, equal parts of alcohol, ether, and fluid may be employed; this favors the separation of the bacteria still more.

Animal Experimentations. The tubercle bacillus can usually be demonstrated in the feces in about 46% of clinically tuberculous cows. In examining the feces of cattle microscopically for tubercle bacilli, however, it should be remembered that there may always be an element of doubt, because saprophytic, acid fast bacilli are always present.

(D) **Tubercle Bacilli in the Bile and Gall Bladder.** In the liver the anatomical picture of tuberculosis is usually that of the miliary form, a tuberculous hepatitis. In the gall bladder tuberculosis is usually a chronic, ulcerative, tuberculous cholecystitis, the result of irritation by stone. The lodgment of the tubercle bacillus in the mucosa of the gall bladder is a secondary affair from tuberculous foci in other organs. If tubercle bacilli do not circulate through the blood stream then they can not be excreted through the bile and gall bladder, tuberculosis is then an impos-

[3]The above method, given by Strassburger for removing tubercle bacilli from feces by the use of alcohol, can be applied to other fluids with equal satisfaction. In urine, both albuminous and albumin free, the use of alcohol is indicated. In urine containing much epithelial cells and pus germs, which may be considered heavy bodies, if centrifuged, the bacilli will, during sedimentation, be carried down into the tube. If to such urine before centrifuging a double volume of alcohol is added, all cellular elements and bacteria as will be more rapidly deposited. To spinal fluid (Liquor Cerebrospinalis) the addition of alcohol precipitates the albumin content in a fine flocculent form. This undoubtedly favors the removal of all cellular bodies, or rapid sedimentation. In pleural exudates the albumin content is usually so great, the albumin sedimentation so enormous, that the bacteria for future demonstration would become lost; here the dilution of the exudate with equal parts of distilled water is advisable before adding alcohol. When desirous of testing any other fluid, even sputum, for tubercle bacilli, it may be treated similarly, using absolute alcohol as a diluent to lower the specific gravity.

sibility—Joest's views are somewhat different. From extensive animal experimentation and study he concludes that tubercle bacilli enter the gall bladder from primary foci in the liver through the bile ducts. The tuberculous processes are not in the tubules but near them and so they become enlarged, break through, their contents becoming mixed with the bile. He maintains that this is similar to tuberculous disease of the female breast where the tuberculously diseased milk glands pour their contents into the ducts and contaminate the milk.

Maxson (1910) questions if tubercle bacilli are ever excreted through the bile and concludes, from personal observation in 12 cases supplemented by 20 cases by Rosenberger, as follows. (97) That while the circulation of tubercle bacilli in the blood and their excretion in the bile is not proved, the evidence is sufficiently strong to make it very probable. (Since then it has been proven that tubercle bacilli very often circulate in the blood.) These 32 observations proved that no matter what the clinical diagnosis in the case was, if autopsy revealed the presence of a tuberculous lesion in either lung, pleura, or peritoneum, the acid fast bacilli could be demonstrated in the bile.

Maxson's technic is as follows:

(1) Add four parts of 15% antiformin solution to one part of bile. Allowed to stand about fifteen minutes in incubator is preferred.

(2) Add equal parts of distilled water. Shake well.

(3) Add ligroin to a depth of about 10 mm.

(4) Allow to stand until sharp separation occurs.

(5) From the turbid zone which appears just below the ligroin transfer ten loopfuls to a clean warm glass slide, allowing the ligroin to evaporate between each loopful. Put the loopfuls on the same spot on the slide.

(6) Fix by heating slide, wash well, stain with carbol-fuchsin, decolorize with acid alcohol, counterstain with methylene blue, wash, dry, and examine.

(E) **Tubercle Bacilli in Exudates and Other Body Fluids.** The demonstration of the tubercle bacillus in exudates, transudates, spinal, meningeal, peritoneal, etc., fluids, does not differ very materially from the procedures employed for the identification of bacilli in milk, urine, etc. The usual methods in all are by sedimentation, concentration, centrifugalization all of which have been given somewhat in detail in this chapter under special

headings to which the student is referred. In the part on Inoscopy (also this chapter) the method of sedimentation is fully described; this may also be successfully employed in searching for bacilli in any of the various body fluids.

The Urochrome or Coloring Matter of the Urine in Tuberculosis.(68) The normal yellow coloring matter of the urine is known as urochrome, the derivative of which appear as two separate substances, one of which can be separated from the urine by lead acetate; the other is not so separated, therefore, they are not identical. These substances are designated urochromogen (A) and urochromogen (B) and by oxidation are changed to urochrome. Both urochromogen A and urochromogen B are proteinoid substances or protein derivatives, oxidation products from the destruction of protoplasmic albumin. Of these two precursors of urochrome, urochromogen A can, by means of potassium salts be oxidized into urochrome; the other, or urochromogen B, if present, gives the Ehrlich or diazo reaction which is dependent upon the presence and amount of urochromogen in the urine; one is identified in the freshly voided urine, the other develops after the urine is kept for 24 hours in the incubator.

Urochrome results from albumin changes, but the presence of its precursors in greater amounts is evidence of toxic destruction going on in the organism. In the tuberculous individual, in the urine of which urochromogen can be demonstrated, it shows that the reduction of organic albumin is not only quantitatively increased, but that it deviates qualitatively from the normal, hence an increased urochromogen in the urine is, in every sense, of toxic origin, and here the toxins of bacterial origin play the leading role. The influence of these toxins is manifested by a destructive condition of the normal oxidative processes going on in the urine, leading not to urochrome, as in the normal process, but to the production of the lower oxides of urochrome.

In 1910 Moriz-Weisz called attention to a new reaction in the urine in connection with the well-known diazo or Ehrlich's. It is designated as the urochromogen, or better as the permanganate test, and is based upon the original Ehrlich's, upon the presence of urochromogen B but acts more promptly than the diazo, reacting with urochromogen A, with which Ehrlich's reacts only after the urine is kept in a moderately warm place for 24 hours. Urochromogen A is only a lower oxide leading up to

the formation of urochromogen B. As the diazo only gives the reaction with the latter, and not with the former, the permanganate test is quicker and more readily positive as it gives the reaction with both chromogens. The technic for demonstrating the permanganate reaction is very simple and more sensitive than the diazo reaction. How this reaction is really brought about is still in dispute. To fresh urine in a urine glass add twice its volume of distilled water, fill two equally sized test tubes about ¾ full, to one add three drops of 1:1000 permanganate of potassium solution; the other is kept as control. By comparing the two test tubes on holding them up to the light, the difference in the color reaction becomes readily apparent. If now a few more drops are added the color may be greatly intensified. Should the urine contain much bile pigment, bilirubin, etc., it is advisable to add a quantity of ammonium sulphate before applying the permanganate test. In the examination of a great number of specimens of urine, both normal and pathological, the permanganate test has been considered a specific in proving the presence of the lower oxides of urochrome.

From numerous observations made by clinicians it is now the consensus of opinion that, especially in pulmonary tuberculosis, a positive urochromogen reaction shows an acutely destructive lesion or process going on in the organism and that a negative reaction in positively pulmonary cases appears to indicate at least, a temporary cessation of all activity. In ordinary chronic, non-active, fibroid cases the reaction is negative and becomes again positive upon a re-exacerbation of the tuberculous process or upon the entering of a new acute intercurrent disturbance. The freshly voided urine from an active tuberculous individual will give a negative diazo; if, however, the urine is placed in an incubator for 24 hours it often becomes strongly positive. The tuberculous organism is incapable of producing the urochrome which the healthy body produces, but in its place it produces a lower oxide, an incompletely oxidized substance, which in all probability is a form of peptonoid or a polypeptonized body unchanged and which in a warm place in 24 hours is oxidized to the higher urochromogen B form.

Roy C. Heflebower,(166) the first in this country to test the value of the permanganate of potassium reaction on the urine, observed the test on 39 tuberculous individuals in tuberculosis sanatoria and arrived at the following conclusions: (1) The fre-

quency and constancy of the diazo and urochromogen reaction in the urine are proportional to the severity of the disease; a constant negative reaction indicates a mild case, whereas a constant positive reaction is evidence of a severe and serious disorder. (2) The urochromogen reaction is more frequent, more constant, than the diazo, therefore, a better index. (3) The intensity of both reactions proves, when constant, a severe tuberculosis.

Corper, Callahan, and Marshak (160) reported their observations before the National Tuberculosis Association in 1916 on 350 patients at the Municipal Tuberculosis Sanatorium of Chicago representing the three stages of the disease, and they concluded as follows:

(1) Cases dying from pulmonary tuberculosis give a positive diazo and urochromogen test at some time during the last six months of their illness. Whenever both tests are negative during this period, death is found to be due to some intervening condition, such as pulmonary hemorrhage, tuberculous meningitis, etc.

(2) Cases of chronic, fibroid tuberculosis generally give both reactions negative, except when same is explicable by some acute exacerbation, acute intercurrent infection, or acute pleural effusion.

(3) In active cases of pulmonary tuberculosis when both reactions are positive and remain so for most of the succeeding examinations, it is of grave prognostic import. When both reactions are negative in active cases no stress can be laid upon the findings.

(4) There seems to be no regularity between the presence of either reaction, its disappearance to be displaced by the appearance of the other or by both.

(5) The presence of a urochromogen reaction in cases showing no clinical symptoms is of no prognostic value. No diazo reactions were obtained in clinically inactive cases, whereas a number of urochromogen reactions were obtained in such cases.

Lime Elimination in Tuberculosis. The increased lime content in the urine of the tuberculous. Demineralization. Decalcification. According to the elaborate studies of Senator, as early as 1877, the urine of the tuberculous contains relatively more lime salts than that of healthy individuals. He states it to be a positive fact that in pulmonary phthisis an abnormal quan-

tity of calcium is excreted in the urine. Robin contends that with the very beginning of tuberculous disease the affected individual shows an increased demineralization, that the increase of lime salt elimination can be of great diagnostic value in the pretuberculous stages of the disease, that is while prodromal symptoms only are present. Sobatta, 1911, states that demineralization may be considered an acquired disposition in the pregnant and the diabetic, hence there is a heightened tendency to tuberculous disease; and that a lime salt reduction in the organism exists more or less in all cases of pulmonary tuberculosis. This creates the tuberculous disposition, hence, therapeutically, lime salts are indicated. It is believed by most physiologists (101) that the lime content of blood serum of healthy individuals is always maintained at a constant level, that in the human blood the variation is from 9 to 10 mg per 100 cubic centimeters.[4]

A. C. Crofton contributed a very interesting paper on the "Increased Urinary Calcium Excretion in Tuberculosis," before the Sixth International Congress on Tuberculosis. He contends that in considering the calcium elimination we must distinguish between that which is eliminated by body changes, the endogenous calcium, and the lime which is brought to the body by way of foodstuffs, the exogenous, both of which are eliminated through the blood and kidneys. The calcium elimination from the body changes is usually constant, does not fluctuate very much from day to day, while the exogenous calcium excretion is inconstant, fluctuates much from day to day, is dependent upon the amount of lime taken in with the food and that but little of this lime is eliminated in the urine, the greater bulk passing out with the feces. To check up the excreted lime from body changes and that from the ingested foods, his examinations were conducted on dogs that were injected with live tubercle bacilli, then fed on food, the accurate calcium content of which was known. Comparing the increased calcium content in the urine after deducting the quantity of lime passed with the stools, he was able to observe a steady lime starvation. From the time of inoculation to the death of the animal, a progressive increase in calcium output was shown. He then made similar observations on the human. In advanced cases of tuberculosis with destruction of

[4] I am not aware that similar observations have been made on the tuberculous to see if this equilibrium is here also maintained; if not, it may in part account for the lime salt starvation in these patients.

lung tissue the urinary excretion of calcium is markedly increased, the figures are always higher than in the normal.

The quantitative determination of calcium in the urine according to McCruddin (16) is as follows. The urine if alkaline must be made neutral or slightly acid with hydrochloric acid and filtered or if turbid or faintly acid add 10 drops of pure HCl. Two hundred cc of the clear acid urine are treated with 10 cc of a 2.5% oxalic acid and 8 cc of a 20% sodium acetate solution. Allow the mixture to stand over night at room temperature or shake vigorously for 10 minutes. Filter off the precipitated calcium oxalate and wash it cl-free with 0.5% ammonium oxalate solution. Save the filtrate if desired for the determintion of magnesium. The precipitated calcium oxalate is then dried on filter paper at 100° C, is placed into a weighed platinum crucible, burned over a bunsen flame until the filter paper is completely ashed. The blast-lamp is then applied and the crucible is then dried in a desiccator and weighed. The increase in weight represents the calcium oxide (CaO) in 200 cc of urine. A simple calculation will yield the amount of CaO in the 24 hours specimen.

Cytodiagnosis in Tuberculosis.(89) A study of the character, number, and kind of cellular elements found in pleural effusions, in spinal, peritoneal, endocardial fluids, or in various other serous effusions is known as cytodiagnosis. It, like all other methods of diagnosis, can not be taken singly. It is only in connection with the clinical history and physical findings that it becomes a valuable aid in the study of tuberculosis. An enormous amount of good work along these lines has been done, especially by French clinicians. Widal and Ravaut of Paris offer the following. The examination of a pleural exudate in which certain cellular bodies are present in varying amounts may offer important points in etiology, diagnosis, and pathogenesis. A few cubic centimeters of pleural fluid are withdrawn by aspiration, defibrinated, and centrifuged. (a) In idiopathic pleurisy the exclusive picture is a preponderance of the small lymphocytes with perhaps a few red corpuscles: (b) in tuberculous pleurisy we may find a few deformed polynuclears, but principally lymphocytes; (c) in streptococcus, sero-fibrinous pleurisy the polynuclear-neutrophiles are much in evidence; (d) in pneumococcus pleurisy red blood corpuscles and a few lymphocytes are seen, but the polymorphonuclears are most abundant and a few mono-

nuclear cells of endothelial origin are also present; (e) in traumatic pleurisy and the aseptic pleurisies complicating heart and kidney disease large endothelial cells single and in groups are present.

In tuberculous meningitis there is a constant and perceptible lymphocytosis in the spinal fluid which is pathognomonic of tuberculous disease. Kretschmer (111) reports the case of a soldier who died of tuberculous meningitis in which the tubercle bacilli were demonstrable in the spinal cord, but failed to be shown in the examination of the withdrawn spinal fluid. Here the centrifuged fluid gave a differential count of 75% of small lymphocytes and 25% neutrophiles.

The cellular elements or constituents in the different fluids vary as to the nature of the disease, i. e., acute or chronic. In acute diseases the polynuclears generally predominate, whilst in chronic disorders like tuberculosis the lymphocytes are usually in preponderant numbers, hence a large number of polymorphonuclear cells is indicative of a marked or recent inflammation (acute infection), and a considerable quantity of the mononuclear element, the small lymphocytes, signify either a mild degree and a late stage of inflammation or the presence of a chronic non-inflammatory process (tuberculous). The diagnostic value of cytology may be summed up by the cytologic formula formulated by Widal and Ravaut.

(a) A preponderance of the small lymphocytes in a fluid (exudate) either pleural, spinal, peritoneal, endocardial, etc., means a tuberculous effusion (chronic).

(b) A preponderance of the polymorphoneutrophiles in a fluid means (exudate), that it is of acute and infectious origin (acute).

(c) A predominence of endothelial cells in a fluid occurring especially in sheets or plaques means that the effusion is of mechanical origin (transudate).

Inoscopy. (89) Inoscopy is a method, the object of which is to demonstrate in an exudate or transudate the presence of bacteria, chiefly tubercle bacilli, and here advantage is taken of spontaneous clotting. This method as first suggested by Jousset is most dependable and offers good results if the technic is accurately followed. Spontaneous coagulation or clotting of an exudate takes place usually in a very short time and, at the same time separates from the fluid portion all the morphological elements present. If now this coagulum is redissolved by means of

an alkali or much better, as is now done (artificially digested) by means of a pepsin solution, the tubercle bacilli may be readily separated by centrifugalization.

The coagulated mass from 100 cc of exudate requires about 100 cc of the following pepsin solution for digestion:

Pepsin	8.5	grams
Hydrochloric Acid U.S.P.	1.0	cc
Glycerine	10.00	cc
Water q. s. ad.	100	cc

The coagulum is put into a beaker or flask, and the pepsin solution added. It is then placed into a warm room or a thermostat, shaking the mixture occasionally. In 2 or 3 hours complete digestion has taken place. Only digestion of the albuminous matter is accomplished, the tubercle bacilli and the nuclei of the leucocytes remaining intact and the virulence of the bacteria but little affected. From the sediment in the centrifuge tubes, smears are prepared and stained in the usual way. Inoscopy is equally applicable in the examination of the blood; even the sputum from the suspicious tuberculous may be so examined. To fluids which may be suspected of containing tubercle bacilli but which do not coagulate spontaneously, a coagulable substance such as horse serum may be added to remove the bacteria, and sputum, urine, bile, etc., may all be most satisfactorily examined by this method.

MISCELLANEOUS

We shall here first consider a few such topics that in some way are related to the various questions of Tuberculosis. Chapter XXXIX gives the calory values of the different foods, the carbohydrates, fats and proteins and includes a dietary table; Chapter XXXX, the definitions of such words, terms and phrases as are now in general use in the conversation and in the literature of Tuberculosis. Next, a table showing the normal standard weights at the different ages from 18 to 55 inclusive as compared with the height, and a table for making the Standard Tuberculin Dilution is appended. This is followed by an index giving the names of authors with page index, by a bibliographic index and closing with a general index or index of subjects.

CHAPTER 39

CALORIES

Calories, the Food Values of Proteids, Fats, and Carbohydrates as Expressed in Heat Units.

All humans in health and in disease, must be supplied with certain quantities of food to keep up the body's existence. Food is the fuel which the body requires to maintain its weight and its energy. Food is to the body what fuel is to the boiler, and like it, is consumed in producing heat and force. (31) (32)

Generally speaking, in the human, the fuel, necessary to produce force and energy, to sustain life, is measured in units of heat—this is spoken of and known as calories. These heat units or calories may vary in quantity or amounts according to the size, age, activity, work, etc., of the individual. One group or class of foods may be much more in demand by the organism at a given time in life than another; for instance, carbohydrates are much more essential in child life, whilst during the energetic and active period of adult life, proteids as a rule are the more necessary elements of food. The active and the ambitious require more calories per day than the indolent and the lazy; more calories are consumed during the cold than during the warm days. Age and sex also play most important roles. In early infant life the number of calories required by the male and in the adolescent, when the male usually becomes more active than the female, there may be a difference of more than a thousand calories per day. The average daily requirement in men being about 3500 calories as against 2500 in women.

By calorie[1] is understood the amount of heat which is required

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[1]In estimating the number of calories necessary for the body's upkeep we must also consider certain undetermined chemical substances, in all probability labile albuminous, known as vitamines. The presence of these bodies is most essential, their absence from food products is prone to lead to various disorders and diseases like Rickets, Beri-Beri, Xerophthalmic, etc. What effect the presence or absence of vitamines may have on the tuberculous organisms is still somewhat speculative. We must, nevertheless, concede their great importance in the treatment of disease when we consider their significance to the normal and vital functions of the body. Up to the present nothing definite is known concerning these bodies. However, we know that their presence is necessary to the growth and development on the one hand and to the keeping of the body in a healthy condition on the other. Vitamines are of two kinds (or perhaps three), a fat soluble which is necessary to body growth and a water soluble, which possesses antagonistic properties like antineurotic or antiscorbutic, etc. In foodstuffs these bodies are readily destroyed by long cooking, in pickling, in frozen meat, in shredded grains, like in rice, keeping food in salt brine, etc.

In estimating a dietary for the tuberculous these bodies should receive consideration and foods which require long cooking, or pickling, should be excluded and the use of raw fruit and vegetables and milk, nuts, eggs, etc., be encouraged.

to raise the temperature of one gram (15 minims) of distilled water one degree, and by means of this standard all food stuffs are estimated in heat units, or better, calories. The oxidization or consumption of one gram each of proteids, fats and carbohydrates has yielded respectively 4.1, 9.3 and 4.1 calories. According to the pioneer research work as first elaborated by Carl Voit of Munich and which is still accepted as most competent, a healthy individual of average weight, 70 to 75 kilos (150 to 165 pounds), performing moderate muscular labor, requires about 118 grams of proteids (483.8 calories), 56 grams of fats (520.8 calories) and 500 grams of carbohydrates (2050.0 calories) or a total of 3054.6 calories, or heat units.

Life cannot be indefinitely sustained by one single kind of food. It requires, to maintain body energy and life, that the various food materials, (proteids, fats and carbohydrates) be taken as the body needs them during the various periods of exercise or rest. A proportional daily intake of food in calories to meet these requirements, to sustain the body's vital processes without hindrance, is given in the following table. In addition to proteids, fats, and carbohydrates, many mineral salts, water, vegetable and fruit acids, etc., are required to maintain comfortably a proper functioning of the various organs and tissues of the body; coarse food or roughage in the form of green vegetables is necessary to aid in the proper movements of the bowels, lime salts for the upbuilding of the bony structure, water for the blood plasma and tissues of the body, etc.

The table given here shows some of the various food materials consisting of proteids, fats and carbohydrates expressed in calorie values or units of heat per pound, as yielded to the body.

One pound of olive oil yields approximately	4464	calories
One pound of butter yields approximately	3605-3816	calories
One pound of bacon yields approximately	3101	calories
One pound of ham (smoked) yields approximately	1920-2485	calories
One pound of beef (roast) yields approximately	1620-1714	calories
One pound of lamb (chops) yields approximately	1665-1771	calories
One pound of lamb (roast) yields approximately	900-960	calories
One pound of steak (tenderloin) yields approximately	1300-1344	calories
One pound of pork (chops) yields approximately	1752	calories
One pound of mutton (leg roast) yields approximately	1420	calories
One pound of turkey (roast) yields approximately	1295	calories
One pound of chicken (roast) yields approximately	869	calories
One pound of chicken (fricassee) yields approximately	855- 907	calories
One pound of fish (bluefish cooked) yields approximately	670	calories
One pound of fish (Spanish mackerel) yields approximately	715	calories
One pound of fish (salmon) yields approximately	915- 950	calories
One pound of oysters yields approximately	230- 250	calories
One pound of eggs (boiled) yelds approximately	765- 811	calories
One pound of mushrooms (boiled) yields approximately	1262	calories
One pound of cheese (American) yields approximately	2173	calories
One pound of cheese (full cream) yields approximately	1950-2064	calories
One pound of marcaroni with cheese yields approximately	1531	calories
One pound of cream yields approximately	1282	calories

One pound of milk yields approximately	325- 346	calories
One pound of milk (malted, prepared) yields approximately	300	calories
One pound of milk (skimmed) yields approximately	178	calories
One pound of milk (butter) yields approximately	173	calories
One pound of sugar yields approximately	1968	calories
One pound of potatoes (boiled) yields approximately	440- 466	calories
One pound of potatoes (chips) yields approximately	2827	calories
One pound of potatoes (sweet, boiled) yields approximately	979	calories
One pound of bread (white) yields approximately	1296	calories
One pound of bread (graham) yields approximately	1282	calories
One pound of bread (brown) yields approximately	1050-1109	calories
One pound of bread (ginger) yields approximately	1670	calories
One pound of bread (whole wheat) yields approximately	1140	calories
One pound of bread (rolls) yields approximately	1395	calories
One pound of cake yields approximately	1776	calories
One pound of pie (apple) yields approximately	1270-1392	calories
One pound of pudding (rice) yields approximately	720	calories
One pound of rice (boiled) yields approximately	525- 538	calories
One pound of oatmeal yields approximately	302	calories
One pound of crackers (soda) yields approximately	1925-2035	calories
One pound of crackers (graham) yields approximately	2095	calories
One pound of beans (baked) yields approximately	600- 955	calories
One pound of beans (lima) yields approximately	768	calories
One pound of beans (canned) yields approximately	634	calories
One pound of corn (stewed or green) yields approximately	480	calories
One pound of peas (dried) yields approximately	1644	calories
One pound of honey yields approximately	1608	calories
One pound of prunes (stewed) yields approximately	1675	calories
One pound of raisins yields approximately	1699	calories
One pound of dates yields approximately	1709	calories
One pound of apricots yields approximately	1056	calories
One pound of rhubarb yields approximately	739	calories
One pound of grapes yields approximately	355	calories
One pound of bananas yields approximately	317	calories
One pound of apples yields approximately	307	calories
One pound of figs yields approximately	1560	calories
One pound of current jelly yields approximately	1550	calories
One pound of oranges yields approximately	254	calories
One pound of squash yields approximately	331	calories
One pound of beets (cooked) yields approximately	185- 197	calories
One pound of tomatoes yields approximately	105- 110	calories
One pound of celery yields approximately	91	calories
One pound of lettuce yields approximately	96	calories
One pound of cabbage yields approximately	24	calories
One pound of turnips yields approximately	19	calories

The following list from the Modern Health Crusade appeared in the April, 1920, number of the Bulletin of the National Tuberculosis Association. The calorie values of the various articles of diet as here given are expressed in teaspoonful, tablespoonful, piece, slice, cup, etc., and are especially recommended in the treatment and care of the puny, the sickly, the underfed, the undernourished, and the suspected tuberculous child.

Cereals

		Calories
Rolled oats	1 cup	147
Bread	1 slice	202
Bread with butter	1 slice	230
Bread with peanut butter	1 slice	221
Ginger bread	1 slice	281
Corn bread	1 slice	194
Shredded wheat	1	110
Farina	1 cup	169
Rice	1 cup	180
Cream of wheat	1 cup	165

Cornmeal mush	1 cup	169
Cracked wheat	1 cup	198
Hominy	1 cup	143
Macaroni and cheese	1 cup	302
Barley soup	1 cup	131
Hot biscuit	1	104
Griddle cakes	1	101

Meats

Meat loaf	1 slice	209
Roast lamb	1 slice	333
Hamburger steak	1 slice	138
Beef stew	1 cup	691
Sardine	1	65
Frankfurter	1	180
Fish chowder	1 cup	372
Brown gravy	½ cup	36
Roast beef	1 slice	333
Cheese fondue	1 cup	529
Meat balls and rice	1 cup	169
Baked fish	1 slice	201
Pot roast	1 slice	240
Codfish balls	1	88
Corned beef	1 slice	608
Smoked pork	1 slice	827

Vegetables

Boiled onions	1	49
Pea soup	1 cup	279
Escalloped potatoes	2 heap'g tbs.	242
Escalloped tomatoes	1 cup	198
Baked beans	1 cup	385
Beets	1	83
Cabbage salad	1 cup	32
Lettuce	4 leaves	8
Peas and carrots	1 cup	69
Bean loaf	1 cup	547
Tomatoes	2 (raw)	41
Greens	1 tb.	28
Lima beans	1 cup	326
Potato salad	1 cup	143
Lentils	1 cup	269
Baked potato	1	147
Boiled cabbage	3 tbs.	100
Spinach	2 tbs.	57
Boiled potato	1	143
Bean soup	1 cup	505

Desserts

Syrup	1 tsp.	117
Rhubarb sauce	1 tb.	78
Cream toast	1 slice	348
Ginger cookie	1	41
Apple tapioca	1 cup	235
Custard	1 cup	362
Junket	1 cup	208
Cake	1 piece	164
Jelly	1 htsp.	113
Oatmeal cookie	1	115
Fruit cookie	1	110
Fruit cake	1 slice	192
Fruit shortcake	1 slice	350
Cheese	1 cu. inch	90
Fudge	2 pieces	221
Cornstarch pudding	2 htsp.	166
Indian pudding	2 htsp.	225
Steamed fruit pudding	2 slices	569
Bread pudding	1 cup	380

Fruit

Prunes	5	143
Apple sauce	3 tbs.	136
Baked apple	1	120
Baked banana	1	144
Apricot or peach sauce	3 tbs.	218
Dates	10	269
Berries (with 1 tsp. sugar)	3 htbs.	120
Orange	1	75
Banana	1	90

Drinks

Milk	1 quart	695
Milk	1 glass	220
Cocoa	1 cup	126
Coffee	1 cup	00
Tea	1 cup	00

For convenience this supplemental list is added here, giving the food values or calories in ounces instead of in pounds.

CALORIES PER OUNCE

Apricots	13
Parsnips	15
Carrots	13
Celery	5
Onions	16
Turnips	8
Farina	105
String Beans	9
Lima Beans	90
Corn Flakes	103
Spaghetti	103
Cheese	106
Olive Oil	200
Oleomargarine	204
Sugar	106
Molasses	79
Potatoes	18
Eggs	45

Butter Beans	22
Kidney Beans	92
Navy Beans	96
Tomatoes (Cans)	5
Beets	22
Corn	27
Cornmeal	100
Corn Syrup	80
Barley	103
Radishes	5
Peas (Canned)	14
Peas (Split or dried)	93
Lettuce	5
Cauliflower	8
Cabbage	9
Sauer Kraut	9
Parsley	4
Grapes	20
Jelly	50
Peaches	14
Rice	102
Tapioca	99
Raisins	95
Dates	80

Milk	20
Butter	214
Flour	98
Corn Starch	103
Chocolate	130
Bread (Fruit)	75
Bread (Bran)	65
Bread (W. Wheat)	70
Bread (Rye)	62
Crackers (Graham)	103
Crackers (Soda)	118
Oats, Rolled	112
Wheat, Puffed	103
Wheat, Cracked	105
Ginger Snaps	100
Peanut Butter	175
Lard	204
Lamb	54
Beef and Soup Bone	59
Pork fresh (lean)	41
Pork fresh (fat)	78
Salt Pork	222
Bacon	169
Apples	15

CHAPTER 40

DEFINITION OF WORDS, TERMS, AND PHRASES USED IN CONVERSATION AND IN THE LITERATURE ON TUBERCULOSIS

These words and phrases are usually found in the writings on topics on tuberculosis and are used by physicians doing special tuberculosis work; in general medicine they are but little used. The medical student interested in this problem should familiarize himself with these terms so as to be able to use them properly. It is most painful to the ear when a speaker (or perhaps a writer) refers to a tubercular disorder when he really means a tuberculous condition. The following words, terms, and phrases are now in general use. (9) (10) (11) (12) (54) (55) (56)

Tuberculosis, Tuberculous and Tubercular. These three words have their origin in common and are derived from the same root; namely, from tuber or tubercle, a small swelling or nodule.

The word tuberculosis (from tubercle, a small nodule and osis, a morbid condition) is now generally recognized as referring to a pathologic process or a disease somewhere in the human body which is produced by the tubercle bacillus. We are all agreed as to when to use the word tuberculosis; that is, when we refer to the disease entity produced by the Koch bacillus; but we are not all in harmony as to when we should say tubercular or when tuberculous. A lesion found anywhere in or about the body and which if represented by small nodules, is said to be tubercular. Cruveilhier, as early as 1862, produced small nodules in the lungs, liver, etc., (see Chapter 1) by means of minute droplets of mercury; and particles of dirt, fabric, silica, etc., often produce in the human body, especially in the pulmonary tissue, similar minute bodies. Even the ova of the tape worm are frequently found imbedded in the abdominal viscera producing similar small nodules. These are all correctly described as tubercular nodules or as tubercular tissue. Leprosy is a tubercular disease; small nodular masses are found in and about the cuticle brought about by the leprosy bacillus. Even nodular masses produced in the lungs by the presence of the tubercle bacillus may rightly be

called a tubercular affair if we do not know their source. Hence anything on or within the body in the form of small nodules may with propriety be referred to as a tubercular condition. If, however, tubercles or small nodules are brought about by the presence of the Koch bacillus, then by common usage, we make a distinction; that is, if we know that the pathological process anywhere in or on the body is brought about by the tubercle bacillus, then it is correct to refer to it as a tuberculous affair.

Briefly, then, when we speak in a general sense of nodular masses in or about a tissue or organ, we should say tubercular; but when we allude to or speak about the disease entity brought about by the tubercle bacillus, that is, when we speak in a specific sense, then only is it correct to say tuberculous, and wrong to speak of it as tubercular. Tuberculous is the adjective applied to lesions regardless of their form, caused by the tubercle bacillus; tubercular, the adjective which is applied broadly, includes every condition and describes the appearance of the lesion regardless of its etiology.

Tuberculously infected and tuberculously diseased. These expressions are very frequently used synonymously. They are, however, distinct, have separate and very different meanings. Generally speaking, we say that we are all infected, but few are tuberculously diseased. The tuberculously infected individual gives evidence that at some time in life he came in contact with the virus, that is, with the tubercle bacillus; that the bacillus has found a resting place or nidus somewhere in his body, become perfectly contented, made its presence known in the lymph tissue, after which it assumed a more or less passive existence.

The great majority of us go about our affairs in our daily life wholly unconscious that somewhere in our interior, we have a colony or group of these invaders. We recognize today that nearly all are tuberculously infected and that people living in large congested districts in cities are all infected. By means of the diagnostic tuberculin test we can prove the existence of these micro-organisms and their toxins in our bodies. If we go about our affairs in a hygienic, careful and healthy way, as is the rule in the majority of individuals, we never become cognizant of this infection. Should we, however, become careless about ourselves, as so frequently happens in early adolescence, with late hours, indulgence in drink, excesses of all kinds with fatigue or exhaustion (even too much football, baseball, golf, dancing, swim-

ming, etc.) with insufficient rest for recuperation, then the body lowers the barriers which for years perhaps have kept the invaders within their confines, and we find that the individual in addition to being tuberculously infected is now also tuberculously diseased.

In the tuberculously infected, as a rule, there are no symptoms except perhaps now and then a few enlarged glands; in the tuberculously diseased, symptoms usually play a leading role. **Briefly**, then, we may state that every body is tuberculously infected, approximately 100%. Of this number about 60% go through life unaware of the infection and about 30% at some time during their existence become also tuberculously diseased. In these, with care, treatment, and obedience to the principles of hygiene, the disease becomes again arrested and they then again remain only tuberculously infected; in the remaining 10% the infection is followed by active tuberculous disease and this class remains throughout life not only tuberculously infected, but chiefly tuberculously diseased.

The open and the closed cases of pulmonary tuberculosis. An individual who presents himself for examination and who gives a history of some previous pulmonary disorder, pneumonia, pleurisy, etc., perhaps months or even years ago, of having lost weight, with poor appetite, with cough at times, slight temperature, clinical findings positive, and with tubercle bacilli in the sputum would be designated an open case of pulmonary tuberculosis. Should, however, all the above mentioned signs and symptoms, with positive clinical findings be present, but with repeated negative sputum findings, then the case in all probability would be called a closed case of pulmonary tuberculosis.

In the so-called open cases, identical with the infectious, there is a free communication from the diseased area in the lungs to the outer atmosphere; the necrotic tissue and bacilli have an outlet into the bronchial tubes and are expectorated with the sputum. These, known as the open or sputum positive cases, become the chief source of spreading infection and disease, and are the highly dangerous ones. In the closed or sputum negative cases, although the physical findings are positive or perhaps only slightly positive and which are usually the more or less slow cases, there exists no communication between the tuberculous area and the outer world, the tuberculous tissue is still walled off, and although there may be cough and expectoration, tubercle

bacilli are not present in the sputum. Such individuals are not dangerous to any community so long as they remain closed or sputum negative, but the examining physician must always bear in mind that closed cases may at any time become open; hence in all such cases it becomes the imperative duty of the examining physician to make at least one sputum test every thirty days. **In short,** then, the open cases of pulmonary tuberculosis are those in which the sputum is tubercle bacilli positive; in these there is a direct communication between the tuberculous area in the lung tissue and the outer atmosphere. The closed or sputum negative cases are those in which the physical findings may or may not be positive; here the pathologic area in the lung appears to be walled off and as yet there is no free communication with the bronchial system.

Phthisis and Tuberculosis. A very fine distinction in the use of these two words is often made. Some maintain that phthisis refers to the chronic form of the disease and tuberculosis to a more acute, a more recent affair. The word "consumption," so much in use by the laity, is undoubtedly derived from the picture seen of the phthisical, and not of the tuberculous individual. The patient with marked cachexia, pronounced emaciation, fixed and immovable chest, prominent ribs, much loss in weight, skin pale and dry, with a facial expression which denotes difficult or labored breathing, is said to be a phthisical subject, but a patient, regardless of how long the disease has existed, who is suffering from chronic pulmonary disorder and does not show much disturbance, has a fairly good appetite, no less in weight or perhaps very little, and is able to be about, perhaps following his avocation, is said to be tuberculous. If a patient is running a temperature, pulse rapid, cough pronounced, expectorations free, many bacilli in the sputum, with ulceration and destructive processes going on in the lungs, with definite cavities, then we should say that such a patient is phthisical. On the other hand, if we should find one or both apices involved, localized rales, temperature not very high, pulse about 100, with little or no evidence of lung destruction, we say that such a person has tuberculosis.

Note the distinct difference in the use of these words in pathology. If in cutting through a lung or kidney we find many cavities, much tissue destruction with pus and caseous material, we refer to it as a phthisical lung or kidney. If, however, at autopsy we find the organs diseased, evidence of a tuberculous

process going on, but with very little or no tissue destruction, and no cavities, we speak of it as tuberculosis of the lung or kidney. Hence, the severity of the disease, regardless of duration or chronicity, tells us when to say phthisis and when tuberculosis.[1]

Sanitarium and Sanatorium. Sanitarium (from sanitas, health). A health station; a place or institution where the conditions are such as especially to promote health and vigor. The word is often incorrectly employed for sanatorium (from sanare, to heal), which is a hospital or place for curing those who are sick, especially a private hospital. Gould's Dictionary.)

Sanitarium, an establishment for the treatment of diseased persons, especially a private hospital for convalescents or those who are not extremely ill.

Sanatorium, from sanatorius, conferring health.

(Dorland's Medical Dictionary.)

Sanitarium, (sanitary; L. sanitas, health). A health station or retreat. Sanatorium, from **sanatary.** (sanatorium, L. Sanare, to heal): an establishment for the treatment of the sick, a rest for invalids. **Sanitary** and **sanatary** should not be confused. **Sanitary** has the more general meaning of pertaining to health, while **sanatary** signifies conducive to health. (Webster .unabridged.)

These are the definitions of the words "Sanitarium" and "Sanatorium" as found in our standard dictionaries. There exists still a great deal of uncertainty as to when to use the one and when, the other. It is becoming more and more accepted that when we refer to an institution for convalescent patients, a rest cure, a retreat, or a place where those are treated who are suffering from a nervous breakdown or from mental disorders, etc., we speak of a **sanitarium,** whilst those who are suffering from pulmonary tuberculosis, in the beginning or in the moderately advanced stage, and who desire to be hospitalized, are said to be assigned to a **sanatorium.** There are, however, exceptions to this rule which cannot always be altered. For instance, the large institution in the City of Chicago for the care of the tuberculous is known as the Municipal Tuberculosis Sanitarium. The word

[1]In this connection, it may be worthy of note to state that many well-known lung specialists make very little, if any, distinction in the use of these terms, using the word "phthisis" in a broad and general sense. They assert that any lesion in the lungs, regardless of size, or amount of involvement, if brought about by the Koch's bacillus should be spoken of as a phthisical process and that the words "consumption," "tuberculosis," "scrofula," etc., have no special meaning, that they all refer to one and the same condition in the lungs, and that is "phthisis."

"sanitarium" was written into the bill, permitting the community to erect such an institution, at the time it was presented before the legislature in Springfield for passage. After its passage, it was not deemed advisable to make the change from "sanitarium" to "sanatorium," owing to the fact that the change might affect the bill as it stood, so its promoters allowed it to stand. It should, however, read "Sanatorium" and not "Sanitarium," to be in accordance with common usage.

The use of the word tubercular in connection with a sanatorium, or a hospital, is not good language, and should not be used; there exists no such thing. To say "tubercular sanatorium," "tubercular hospital," does not mean an institution where patients suffering from pulmonary tuberculosis are being taken care of, but it signifies a building or structure on or in which nodular masses are being developed or growing.

Ambulant, Ambulatory. From **ambulant,** to walk; conveying; pertaining to walking.

These words are very frequently used when referring to tuberculous patients who are not suffering from far advanced disease. A patient who is suffering from pulmonary tuberculosis and is being treated at the physician's office, at a tuberculosis clinic, or dispensary, i. e., who is in a condition to come for treatment, is said to be an ambulatory tuberculous individual, whilst a patient whose condition does not permit him to come for assistance, but who must be taken care of at the sanatorium or his home is spoken of as a stationary, home, or bedfast patient. So long as the patient's pulmonary condition remains quiescent and he is able to walk about, he is ambulatory, but in those cases in which the disorder progresses, the patient passes automatically into the bedfast or non-ambulatory class.

Hilum pl. Hila and Hilus, Hilar, Etc. A difference of opinion exists amongst clinicians and teachers concerning the correct use of these words. By the term Hilum we designate the depression, pit or recess observed in some organs, that is where blood vessels, etc., enter and leave the organ, as for instance, the hilum of the lung, kidney, liver, spleen, etc. Many writers and teachers maintain that the word Hilus has no place in literature in referring to these depressions and that the word hilum only should be used. Some dictionaries, for example, Gould's, describes the word Hilum but does not mention the term Hilus. Both words are now so freely used in the

literature on tuberculosis that to both a distinct and definite place must be assigned. When referring to the depression or when used in the sense of a noun, say Hilum, like "the hilum of the lung," "the liver," etc., but when used in a generic term or as an adjective, say Hilus, like hilus tuberculosis, hilus disease, the hilus glands are enlarged, etc., plural the hilar shadows, etc.

Allergy and Anergy. Allergy (opposite Anergy) or Allergie (opposite Anergie), Anaphylaxis (opposite Ananaphylaxis), Hypersusceptibility, Sensibilitrice, Protein Sensitization, The Theobald Smith phenomenon, etc., are all more or less similar terms, expressing a similar phenomenon.

In the chapter on Tuberculin (see Chapter XXIV), reference was made to the experimental work done by Koch. We may briefly refer here to these observations. When an animal (or a human) is injected with a body foreign albumin, in not too great an amount, no obvious effect is observed. If, however, after a given interval a second injection of the same albumin, even in a less quantity is administered, a severe reaction is noticeable, which may be so severe as to cause death in a comparatively short time. This reaction is referred to as shock, as anaphylactic shock, or simply as anaphylaxis. The first injection sensitized the animal to that particular albumin and the reaction is specific for that and no other. To this reaction, Richet (1903) applied the name "anaphylaxis" (from ana-phylaxis—against—protection). This term is not well chosen as it does not express the phenomenon of sensitization or hypersensitiveness.

Later when v. Behring discovered the hypersensitiveness to toxins and accurately described the phenomenon, he suggested the name allergy. The word "allergy" signifies that the body, after coming in contact with a foreign albumin undergoes a change, and becomes sensitized to a subsequent contact with that same albumin. This phenomenon is observed in the tuberculously infected individual who, harboring in his organism a foreign albumin or protein (tuberculin), reacts when he comes in contact with a similar protein through either the skin or mucous surfaces. This is a specific reaction.

Tuberculin. (A) The positive and negative reaction. When tuberculin is applied, diagnostically, to an individual who previously has come in contact with the tuberculosis virus or perhaps is already slightly tuberculously diseased, a reaction is manifested at the point of application by the appearance of an area

of hyperemia. The results are identical if the medicament is applied either to the mucous membrane or to a cutaneous surface, and the conjunctival or Calmette, the percutaneous or Moro, the cutaneous or v. Pirquet, and the intracutaneous or Mantoux are all, if the application proves positive, followed by a more or less reddened zone, indicating that the reacting individual some time in life came in contact with the tubercle bacillus. We designate this as a positive reaction. If, however, in a suspected individual the repeated application of tuberculin is not followed by a reaction, no reddened area, no systemic disturbance, then we say that the tuberculin test is negative. This, however, should not always be construed as signifying that the tested individual has never come in contact with the disease producing germs, because in advanced cases of pulmonary tuberculosis, and in recent infections in which the newly infected organism has not been called upon to develop defense agencies, the tuberculin application is generally always negative.

Briefly, then, when tuberculin is used for diagnostic purposes, and its application is followed by an area of redness, it is said to be positive; if not, negative. Hence, we speak of a positive or a negative tuberculin reaction or test.

(B) The positive and negative phase. On the other hand, if tuberculin is given for curative purposes, therapeutically and not diagnostically, and if during the course of treatment, the patient evinces a feeling of well being, becomes much improved, his tuberculous process seems to be arrested, the activity becomes less (evidence of an immunizing response) we designate such findings as the positive tuberculin phase. If, however, during such a course of treatment, or perhaps if the therapeutic dose given produces untoward results such as a slight increase in fever, some malaise, nausea, or aggravated clinical symptoms, or after long use of tuberculin, no improvement in the clinical picture is noticeable, does not seem to influence the disorder in any way, then we allude to this as the negative tuberculin phase.

In short, when tuberculin is used for diagnostic purposes we speak of either a positive or a negative reaction; if, however, given therapeutically, we observe its effect on the tuberculously diseased individual and speak then of a positive or a negative phase.

Manifest Tuberculosis. (Evident tuberculosis.) This expression is very much in use in foreign literature and is now used by

many phthisio-therapeutists. This term is usually applied when, in a given case of pulmonary tuberculosis, the conditions are such that by means of a chest examination the physical signs can be demonstrated to be positive. It indicates that the tuberculosis is active.

Latent Tuberculosis. This is the direct opposite of manifest tuberculosis. In writings on tuberculosis, it is also often referred to as hidden, obscure, occult, pretuberculous, etc. It expresses an inactivity of the disease, and is said to follow after infection, the infection remaining quiet or latent, not active, until some body disturbance takes place when the infection is followed by manifest disease. So long as the infection remains as it is, presenting no symptoms or signs of activity, it is called latent.

Clinical Tuberculosis. When both subjective and objective symptoms are present, and when by physical examination the disease is demonstrated, it is referred to as clinical tuberculosis. If, in the examination of the chest, the physical signs are obscure, not definite, the diagnosis doubtful, then it is not spoken of as clinical tuberculosis. Clinical tuberculosis may or may not be active, but the physical signs are positive. Hence, we may say that clinical tuberculosis is tuberculosis with or without activity; manifest tuberculosis, tuberculosis with activity; and latent tuberculosis is suspected pulmonary tuberculosis in which the lesion is not clearly demonstrable by physical examination.

Tuberculous Lesions. This denotes the physical changes which have taken place in the lungs after infection. It usually refers to the spots or areas in the lung which are now tuberculous, are now abnormal or infiltrated.

Immunity and Virulence—see Chapter 9—Immunity.

Infection—see Chapter 5—Infection and Contagion.

Subjective and Objective Symptoms—see Chapter 11—Symptomatology.

The definitions of the words "quiescent," "arrested," "cured," etc., are here fully given in the definition of the terms, "incipient," "moderately advanced," and "far advanced," as suggested by the National Tuberculosis Association.

Terms Used in Definition of Incipient Tuberculosis.

1. **Slight Constitutional Disturbance.** Slight loss of appetite, of strength, of weight, lassitude; possibly slight acceleration of pulse or possibly slight elevation of temperature. The impair-

ment of health may be so slight that the patient does not look or feel sick in the ordinary sense of the word.

2. **Slight Elevation of Temperature.** Maximum temperature after rest for one hour, never over 99.5 to 100° F. by mouth, (or 100.5° per rectum).

3. **Slight Acceleration of Pulse.** Maximum pulse rate not over 90 after rest for one hour, sitting or lying, except when due to causes other than tuberculosis.

4. **Absence of Tubercule Bacilli or "closed case."** Each monthly examination (if the sputum be negative) should consist of a careful microscopic examination, with a mechanical stage, of two smears, devoting at least three minutes to each smear, made from selected particles (from at least different parts) of the sputum on each of three successive days. The morning sputum should always be obtained, or, better, the minute bits that some arrested patients raise at very infrequent intervals. It is not yet deemed wise to insist on digestion and centrifugalization, or on inoculation of guinea pigs. If bacilli are present, the case is called "open" for the following 30 days.

5. **Infiltration.** Physical signs of slight prominence of the clavicle, lessened movement of chest, narrowing of apical resonance with lessened movement of base of lung, slight or no change in resonance, distant or loud and harsh breathing with or without some change in the rhythm, (i. e., prolonged expiration), vocal resonance possibly slightly increased, or fine or moderately coarse rales present or absent.

6. **Apex.** That portion of the lung situated above the clavicle anteriorly and the third dorsal spine, posteriorly.

7. **A Small Part of One Lobe.** An area of one or two intercostal spaces, or an area not exceeding 6 to 8 cm. (2 or 3 inches) in extent, according to the size of the patient.

Terms used in Definition of Moderately Advanced Tuberculosis.

1. **Marked Impairment of Function, Either Local or Constitutional.** Local: Marked dyspnea on exertion limiting seriously the patient's activity. Constitutional: Marked weakness, anemia, tachycardia.

2. **Moderate Extent of Localized Consolidation.** An area of one-half lobe or less, which may involve both apices; marked dulness, bronchial or decidedly broncho-vesicular breathing; markedly increased vocal resonance; rales usually present. These

signs are apt to be sharply limited as to area instead of gradually shading into normal physical signs.

3. **Evidence of Destruction of Tissue.** Presence of tubercle bacilli or elastic fibers in the sputum, or the presence of the physical signs of a cavity. There are no absolutely certain physical signs of a cavity but a combination of any four of the following signs is to be taken as indicative of a cavity: (1) cracked pot note; (2) amphoric breathing; (3) intense whispering pectoriloquy; (4) a veiled puff or post tussive suction; (5) bubbling or resonant rales. "Physical signs of softening" do not admit of any definition apart from that of cavity formation, and the term should not be used.

4. **Disseminated Fibroid Deposits.** More or less localized areas of fibrous tissue, producing on physical examination some change or dulness in the percussion note, more or less increase of vocal resonance, harsh, suppressed, or broncho-vesicular breathing, rales usually sibilant or sonorous, but at times fine or moderately coarse.

5. **Serious Complications.** These should be limited to tuberculous complications, such as meningitis, pharyngitis, laryngitis, (except slight thickening of the posterior interarytenoid space, and superficial ulceration of a vocal cord), enteritis, peritonitis, nephritis, cystitis, orchitis, adenitis, (unless very slight), etc.

Terms Used in Definition of Far Advanced Tuberculosis.

1. Marked consolidation indicates dulness merging into flatness, bronchial or tubular breathing, and other signs of consolidation as defined above.

Classification of Terms Used Upon the Discharge of Patient.

(a) **Apparently Cured (formerly "cured").** All constitutional symptoms and expectoration with bacilli absent for a period of two years under ordinary conditions of life.

(b) **Arrested.** All constitutional symptoms and expectoration with bacilli absent for a period of six months; the physical signs, those of a healed lesion.

(c) **Apparently Arrested (formerly "apparently cured")** All constitutional symptoms and expectoration with bacilli absent for a period of three months; the physical signs, those of a healed lesion.

(d) **Quiescent (formerly "arrested").** Absence of all constitutional symptoms; expectoration with bacilli may or may not be

present; physical signs stationary or retrogressive; the foregoing condition to have existed for at least two months.

(e) **Improved.** Constitutional symptoms lessened or entirely absent; physical signs improved or unchanged; cough and expectoration with bacilli usually present.

(f) **Unimproved.** All essential symptoms and signs unabated or increased.

(g) **Died.**

Terms Used in Definition of "Apparently Cured."

1. **Constitutional Symptoms (absent).** These include elevation of temperature, loss of weight, loss of strength, night sweats, chills, tachycardia, cyanosis, loss of appetite, amenorrhea, etc.

2. **Physical Signs of Healed Lesion.** These may embrace every physical sign of infiltration or consolidation (see above) with the exception of rales, which must be permanently absent, except possibly a few fine rales at the base, probably atelectatic or marginal in origin, at one apex, or over a small part of one lobe. Rales in the latter two places are to be heard only during the cough, at the end of a prolonged expiration, or late in inspiration which follows the cough.

Terms Used in Definition of "Improved."

Constitutional Symptoms Lessened or Entirely Absent. By this is meant an improvement in the general condition as shown either by a gain in both weight and strength or by reduction of previous febrile temperature at normal without loss of strength.

Terms Used in Definition of "Unimproved or Progressive."

Essential Symptoms and Signs. These include, among others, weight, strength, appetite, night sweats, hemoptysis, pleurisy, dyspnea, temperature, pulse rate, dulness, changes in vocal resonance and respiratory movement, rales.

Terms Used in Definition of "Cured."

Ordinary Condition of Life. This term as used implies that the patient is able to live in an environment where he is able to support himself without the assistance of others, or to live in his former surroundings and pursue his former occupation.

Terms in Definition of "Onset."

Catarrhal, Pleuritic, Insidious, Hemorrhagic, Febrile, etc.

Definition of Term "Temperature on Admission."

Average maximum temperature for the first seven days.

Definition of "General Condition on Admission and on Discharge."

Favorable, Unfavorable.

Definition of Term, "Temperature on Discharge."

Average maximum temperature for the last two days.

Definition of Term "Digestion on Admission and on Discharge."

Impaired or unimpaired.

TABLE OF NORMAL STANDARD WEIGHTS—MALES

Stripped and Without Shoes

Normal Weight of an Individual According to Height and Age

Inches	60 5 ft.	61	62	63	64	65	66 5 ft. 6 in.	67	68	69	70	71	72 6 ft.	73	74	75	76	
Age																		Age
18	108	111	115	118	122	126	130	134	139	143	148	153	157	156	167	172	177	18
19	108	112	116	119	123	127	131	135	140	144	149	154	158	163	168	173	178	19
20	109	113	117	120	124	128	132	136	141	145	150	155	159	164	169	174	179	20
21	110	114	116	121	125	129	133	137	142	146	151	156	160	165	170	175	180	21
22	111	115	117	122	126	130	134	138	143	147	152	157	161	166	171	176	181	22
23	111	115	119	123	127	130	135	139	144	148	153	158	162	167	172	177	182	23
24	112	116	120	123	128	131	136	140	145	149	154	159	163	168	173	178	183	24
25	113	117	120	124	128	132	136	141	146	150	155	160	164	169	174	179	184	25
26	114	118	121	125	129	133	137	142	146	151	156	161	165	170	175	180	185	26
27	114	119	120	126	130	134	138	142	147	152	157	162	166	171	176	181	186	27
28	115	119	122	126	130	134	139	143	148	153	158	162	167	172	177	182	187	28
29	116	119	123	127	131	135	140	144	149	154	159	163	168	173	178	183	188	29
30	116	120	124	128	132	136	140	144	149	154	159	164	169	174	179	184	189	30
31	117	121	124	128	132	137	141	145	150	155	160	165	170	175	180	185	190	31
32	117	121	125	129	133	137	142	146	151	156	161	165	171	176	181	186	191	32
33	118	122	125	130	134	138	142	147	151	156	162	166	171	176	182	186	191	33
34	118	122	126	130	134	139	143	147	152	157	162	167	172	177	182	187	192	34
35	119	123	126	131	135	139	144	148	153	158	163	167	173	178	183	188	193	35
36	119	123	127	131	135	140	144	149	153	158	164	167	173	178	183	188	193	36
37	120	123	127	132	136	140	145	149	154	158	164	169	174	179	184	189	194	37
38	120	124	128	132	136	141	145	150	154	159	165	169	175	180	184	189	195	38
39	121	124	128	133	137	141	146	150	155	160	165	170	175	180	185	190	195	39
40	121	125	128	133	137	141	146	150	155	160	165	170	176	180	185	190	196	40
41	121	125	129	133	138	142	146	151	156	160	165	171	176	181	186	190	196	41
42	122	125	129	134	138	142	147	151	156	161	166	171	177	181	186	191	196	42
43	122	126	130	134	138	143	147	152	157	162	167	172	177	182	187	192	197	43
44	122	126	130	135	139	143	147	152	157	162	167	172	178	182	187	192	197	44
45	123	126	130	135	139	143	148	153	157	162	168	173	178	183	187	192	198	45
46	123	127	131	135	139	144	148	153	158	162	168	173	178	183	188	192	198	46
47	123	127	131	135	140	144	149	153	158	163	168	173	179	183	188	193	198	47
48	123	127	131	136	140	144	149	153	158	163	169	174	179	183	189	193	199	48
49	123	127	131	136	140	145	149	154	159	163	169	174	179	184	189	193	199	49
50	124	128	132	136	140	145	149	154	159	163	169	174	180	184	189	193	199	50
51	124	128	132	137	141	145	150	154	159	164	170	174	180	184	189	194	200	51
52	124	128	132	137	141	145	150	154	159	164	170	175	180	184	189	194	200	52
53	124	128	132	137	141	145	150	155	160	164	170	175	180	185	190	195	200	53
54	124	128	132	137	141	145	150	155	160	165	170	175	180	190	190	195	200	54
55	124	128	132	141	148	150	155	160	165	170	175	180	185	190	195	196	200	55

Deduct about 10% from above weight in case of female.

TABLE OF STANDARD TUBERCULIN DILUTIONS AND THEIR EQUIVALENTS

No. 0	Take 1 cc O. T. and 9 cc Na Cl. Solution (phenolized)=No. 0 and 1 cc No. 0=1 in 10 (dg. 0.1 or 0.1)=1 in 10 or a 10% Solution..................................=1½ grain per cc
No. 1	Take 1 cc No. 0 and 9 cc Na Cl. Solution (phenolized)=No. 1 and 1 cc No. 1=1 in 100 (cg. 0.01 or 0.01)=1 in 100 or a 1% Solution...:.............................=1/6 grain per cc
No. 2	Take 1 cc No. 1 and 9 cc Na Cl. Solution (phenolized)=No. 2 and 1 cc No. 2=1 in 1000 (mg. 0.001 or 0.001)=1 in 1000 or a 1/10 of 1% Solution...................=1/60 grain per cc
No. 3	Take 1 cc No. 2 and 9 cc Na Cl. Solution (phenolized)=No. 3 and 1 cc No. 3=1 in 10.000 (dmg. 0.0001 or 0.1 mg.)= 1 in 10.000 or a 1/100 of 1% Solution...........=1/600 grain per cc
No. 4	Take 1 cc No. 3 and 9 cc Na Cl. Solution (phenolized)=No. 4 and 1 cc No. 4=1 in 100.000 (cmg. 0.00001 or 0.01 mg.)=1 in 100.000 or a 1/1000 of 1% Solution=1/6000 grain per cc
No. 5	Take 1 cc No. 4 and 9 cc Na Cl. Solution (phenolized)=No. 5 and 1 cc No. 5=1 in 1.000.000 (mmg. 0.000001 or 0.001 mg.)=1 in 1.000000 or a 1/10.000 of 1% Solution=1/60.000 grain per cc

The Diagnostic Dose of Tuberculin

The diagnostic or probationary dose of Tuberculin equals 1/10 cc of No. 1 (0.001) this is approximately 1/60 grain. Giving a less amount than the diagnostic or probationary dose of Tuberculin equals 1/20 cc of No. 1 (0.0005) this is approximately 1/120 grain, etc.

The Therapeutic Dose of Tuberculin

The initial therapeutic dose of Tuberculin usually equals 1/10 cc of No. 5 (0.001 mg.) this is approximately 1/600.000 of a grain. If a smaller dose is desired, then the initial therapeutic dose of Tuberculin usually equals 1/20 cc of No. 5 (0.0005 mg.) this is approximately 1/1.200.000 of a grain, etc.

We see that the diagnostic dose of Tuberculin is really 10.000 times that of the initial therapeutic dose.

Fig. 61. The Road to Health for the Tuberculous, illustrating the six indispensable and necessary factors for the bringing about of an arrest of the Pulmonary Tuberculous disease.

Designed by a patient now suffering from Pulmonary Tuberculosis and who is religiously observing these six rules and that to the letter, confident that by so doing her tuberculous process will become promptly arrested.

For description of these six most important and necessary rules see page 207, Chapter 19, "The Cure of the Tuberculous."

INDEX OF NAMES OF AUTHORS AND CONTRIBUTORS WITH PAGE INDEX

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